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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 12-double-spaced typed pages, including tables, graphs, and photographs. Because of reproduction costs photographs should be kept to a minimum. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

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IN THIS ISSUE

TO ALL CONTRIBUTORS AND RECIPIENTS OF THE PLANT DISEASE REPORTER:
Please note revision in "Acceptance of Manuscripts" paragraph on opposite page.

VIRUS

C. L. SCHNEIDER reports the first occurrence in Maryland of curly top of sugar beet, in August 1958 at the Plant Industry Station, Beltsville; however, the presence of the virus vector (the beet leafhopper) was not established, page 681.

In the summer of 1958 the sugar beet curly top virus was found for the first time on tobacco also, at both Beltsville, Maryland and Oxford, North Carolina, according to HOWARD E. HEGGESTAD and E. LEON MOORE, page 682.

PHILIP BRIERLEY and PAUL LORENTZ compare a non soil-borne virus isolated from chrysanthemum with the tobacco mosaic virus, to which it may be distantly related, page 685.

Results of a limited investigation, by A. G. PLAKIDAS, of a new chlorotic spot of waxleaf ligustrum indicate that the presumed virus disease, although rather widespread, is relatively unimportant and not severe on ligustrums, page 688.

REN-JONG CHIU and WEBSTER H. SILL Jr.'s search for a much needed satisfactory local lesion bioassay host for bromegrass mosaic virus revealed that among the 33 species or varieties tested Datura stramonium was the only plant on which the number of leaf lesions was directly related to the virus concentration in the diseased leaf sap diluted beyond 10^{-2}, page 690.

JOHN R. McGREW discusses the degree of control obtained of movement of mild mottle virus into commercial strawberries grown on the Eastern Shores of Maryland and Delaware, under conditions of extensive isolation and aphid control, page 695.

J. D. PANZER suggests that "poor rooter" clones of alfalfa be tested for infection by the mosaic virus before attempting to establish plants from cuttings, since this virus impairs root development and growth, page 697.

W. J. ZAUMEYER and GRACIANO PATINO describe a new strain of the pea streak virus found in Idaho and compare it with four previously reported pea streak diseases, page 698.

Two years of field tests by J. A. MILBRATH and K. G. SWENSON on the method of transmission of ring spot virus of stone fruits have demonstrated that the virus will spread naturally from infected squash plants to healthy squash plants, although the vector remains unknown, page 705.

The discovery in 1957 of potato virus Y on pepper and tomato in two new areas of Florida -- the East Coast and Immokalee farming regions -- has borne out JOHN N. SIMONS' earlier prediction that in Florida this virus follows a pattern, that is, it appears on tomatoes and peppers about a year after commercial potato production is introduced, page 710.

A whitefly has been established by E. M. HILDEBRAND as the natural vector of sweetpotato feathery mottle virus in Maryland, page 712.

E. M. HILDEBRAND discusses the conditions necessary for the accomplishment of virus infection of epidermal cells of plants, page 715.

FUNGICIDES

As a result of extensive experiments to test the relationship between gallonage and types of applicators used for control of early blight and anthracnose, using leaf prints for determination of fungicide deposition and distribution, W. T. SCHROEDER, G. L. MACK, J. L. BRANN, and W. W. GUNKEL have found that the principle of concentrate application of tomato fungicides is sound, provided the dosage per acre remains constant, page 719.

Of the several new and old materials used by ROBERT E. STALL in comparison trials for control of the damaging bacterial spot of field seeded tomatoes in Florida, Tri-basic Copper Sulfate again gave the best results under mild infection conditions, while copper plus streptomycin was superior for control of more severe infection, page 725.

According to COLIN D. McKEEN, observations and experiments at Harrow, Ontario have shown that a maneb spray applied to tomato and pepper seedlings grown in the greenhouse results in a collar of collapsed epidermal and cortical tissues on the hypocotyl just above the soil line, an injury which does not occur on field plants, page 729.

In fungicide tests conducted in Delaware by D. F. CROSSAN, W. H. JOHNSON, and M. R. SIEGEL for the control of five different fungal pathogens on four different hosts, maneb proved to be the outstanding material for a wide range of vegetables, page 732.

JOHN J. NATTI reports results of 2 years of tests to find a fungicide or fungicide-streptomycin combination spray that possesses either the property of tenacity on foliage or of systemic activity necessary for control of downy mildew of broccoli, page 735.

ROBERT B. MARLATT, HENRY TUCKER, and JOSEPH K. STEWART evaluate sodium hypochlorite and sorbic acid as dips for control of decay in packaged carrots, page 741.

Of the fungicides tested by W. W. WILLIS, C. T. ROGERSON, and W. J. CARPENTER for control of root rot of Croft Lilies, captan applied as a dust directly to the bulb gave better control, in general, than any other material, applied either as a dust or a soil mix treatment, with the exception that for control of the two most prevalent fungi soil mix was superior to dust treatment, page 745.

According to KENNETH H. GARREN, soil surface applications of PCNB cannot be considered either as a substitute for or a supplement to non-dirting cultivation for control of peanut stem rot in Virginia, page 750.

Four weekly spray applications of either streptomycin sulfate or streptomycin nitrate to Burley tobacco resulted in excellent control of wildfire, in the 1958 studies conducted in North Carolina by LUTHER SHAW, G. B. LUCAS, and JOHN H. WILSON, Jr., page 753.

Compound N521 (Mylone) showed the most promise of seven chemicals tested as soil drenches by O. D. MORGAN for control of black shank of tobacco in Maryland, page 755.

GRASSES, CEREALS, AND FORAGE (see also under Virus, Miscellaneous)

The variety Asosan, which has been resistant to all cultures of the wheat powdery mildew fungus hitherto tested on it, is attacked by a strain discovered by H. R. POWERS, Jr., J. F. SCHAFER, and R. M. CALDWELL and tentatively designated as race 2A, page 762.

Germination capacity decreases and mold invasion of stored wheat increases with increase in moisture content, temperature, and time in storage, according to results of studies made by THOMAS D. WYLLIE and CLYDE M. CHRISTENSEN, page 764.

J. ARTIE BROWNING and K. J. FREY theorize that the mode of inheritance of resistance to certain oat stem rust races indicates possession of a previously unknown recessive gene for resistance by eight strains from the World Collection of Oats, page 768.

FRANKLIN A. COFFMAN concludes, from observations on the performance of the progeny from its crosses, that the century-old hexaploid oat Black Mesdag should prove helpful in making both interspecies and intraspecies crosses, page 772.

M. C. FUTRELL, I. M. ATKINS, and C. D. HOBBS review conditions accompanying the unusually severe incidence of diseases on small grains in Texas in 1957 and 1958, with special attention to the factors responsible for epidemics of stripe rust on wheat during these 2 years, page 777.

EARLE W. HANSON notes reaction of seven new varieties of red clover to several diseases common in Wisconsin, page 782.

NEMATODES

Results of soil fumigation experiments with various materials, conducted by W. A. HAGLUND and THOMAS H. KING, demonstrated that parasitic nematodes can reduce yields of canning peas in Minnesota, but further evidence is needed to determine the relationship between nematodes and the development of root rot, page 787.

M. O. MAUNG and W. R. JENKINS studied the effects of infection by root-knot and stubby-root nematodes on nutrition in tomato plants, page 791.

S. A. SHER, F. J. FOOTE, and S. B. BOSWELL describe damage to avocado plants caused by the root-lesion nematode Pratylenchus vulnus, page 797.

M. T. HUTCHINSON and J. P. REED report the finding of the pine cystoid nematode, Meloidodera floridensis, in New Jersey, page 801.

FRUITS AND TREES (see also under Virus, Nematodes, Miscellaneous)

The Fusicoccum canker of highbush blueberry has become more widespread and severe in Massachusetts since it was first found in the State in 1957, according to BERT M. ZUCKERMAN, page 803.

J. F. HOCKEY describes and suggests measures for the control of a calyx-end rot of apple caused by Sclerotinia sclerotiorum, present in sod mulch orchards in Nova Scotia, page 804.

Evidence reported by ROSS W. DAVIDSON, E. R. TOOLE, and W. A. CAMPBELL indicates a possible causal relation of a species of Stereum to the "pecky" decay of cypress, page 806.

CHARLES D. LEAPHART reports that western white pine and several associated coniferous species in the Inland Empire region suffered heavy damage from the unusually dry and hot summer of 1958, page 809.

Phytophthora cinnamomi was determined by LAWRENCE R. SCHREIBER and RALPH J. GREEN, Jr. to be the cause of a dieback and root rot of yew cuttings and plants growing under unfavorable conditions in Indiana nurseries, page 814.

MISCELLANEOUS

NORMAN C. HAYSLIP and ROBERT E. STALL found that in southern Florida less Rhizoctonia soil rot of tomato fruit resulted when grass alone was used in improved-pasture rotations with tomato than when white clover was included with grass in the improved-pasture plantings, page 818.

In greenhouse experiments, ROSARIO PROVVIDENTI and W. T. SCHROEDER demonstrated that Verticillium albo-atrum can infect leaves of tomato and eggplant and that foliage infection sometimes results in systemic invasion of the plant, page 821.

WILLIAM J. STONE and THOMAS W. CULP consider diseases to constitute the limiting factor in castorbean production in Mississippi, page 827.

Phytophthora spp. pathogenic to citrus crops are prevalent in canals and reservoirs supplying irrigation water to citrus groves in California, according to a survey reported by L. J. KLOTZ, PO-PING WONG, and T. A. DeWOLFE, page 830.

L. J. KLOTZ and T. A. DeWOLFE give directions for more accurate determination of the fungicidal value of chemical treatments against species of Phytophthora, page 833.

Brief Notes on Plant Disease Occurrence, page 834: Aphanomyces raphani on red globe radish in Connecticut, by SAUL RICH. Annual ryegrass, Lolium multiflorum, host for Sclerotium rolfsii, by HOMER D. WELLS. Ascochyta hordei on barley in Ohio, by C. W. ELLETT.

Corrections, page 835.

May Weather, page 836.

OCCURRENCE OF CURLY TOP OF SUGAR BEETS IN MARYLAND IN 1958

C. L. Schneider[1]

Before 1957, the occurrence of the curly-top virus, which is transmitted by the beet leafhopper, Circulifer tenellus, was not recorded east of Minnesota (4), Iowa (4), Wisconsin (2), and Illinois (3, 5). Douglass (1), in summarizing the occurrence of the beet leafhopper east of the Mississippi, stated that the insect had been collected in Illinois and Florida. In 1953, Giddings (3) positively identified the virus from sugar beet plants collected in Illinois, Iowa, and Minnesota as the curly-top virus. In August 1957, sugar beets with curly top were observed by G. H. Coons at Van Wert, Ohio, and this observation was confirmed later by H. W. Bockstahler (oral communication). Troutman and Fenne (6) reported the occurrence of curly-top symptoms on tobacco in Virginia in 1958.

In late August 1958, a 4-month-old sugar beet plant with characteristic curly-top symptoms -- vein clearing, leaf curling, and papillate growths from the veins -- was found in an experimental field of sugar beets at the Plant Industry Station, Beltsville, Maryland. Field identification was confirmed later by G. H. Coons. Subsequently, upon critical inspection of the sugar beet area, comprising about 9 acres, 11 more beets with positive curly-top symptoms were found.

Sweepings were made in the vicinity of the diseased plants and in adjacent areas in October by P. W. Oman and E. A. Taylor, Entomology Research Division, but these investigators did not find any specimens of the beet leafhopper. The diseased plants, after having been moved from the field, were trimmed and caged, but no beet leafhopper nymphs emerged. Thus the presence of the beet leafhopper at Beltsville has not been established.

By April 1, 1959, seven of the diseased plants transplanted from the field to the greenhouse had died. The five plants surviving continued to show typical curly-top symptoms.

Since 1934, experimental plantings of sugar beets have been made each year in the vicinity of the District of Columbia. These plantings, consisting of numerous breeding strains, have been closely observed each year by Dewey Stewart and G. H. Coons during the scoring of individual plants and plots of plants for damage caused by Cercospora leaf spot and other diseases. Although these investigators found savoy and other virus diseases of sugar beets, they did not find curly top in this area prior to 1958.

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OCCURRENCE OF CURLY TOP ON TOBACCO IN MARYLAND AND NORTH CAROLINA IN 1958

Howard E. Heggestad and E. Leon Moore

Summary

Records are provided which constitute the first adequately confirmed cases of curly top on tobacco in Maryland and North Carolina. Presence of curly-top virus in the affected plants was confirmed by beet leafhopper transmission studies. Very few diseased plants were found in the fields, being similar in this respect to reports of curly top from three other tobacco-producing States. The possible relation of disease occurrences to known occurrences and long-distance movement of beet leafhoppers is discussed.

Early in August 1958, a tobacco plant with characteristic symptoms (Fig. 1) of curly top was observed in a tobacco-breeding nursery at Plant Industry Station, Beltsville, Maryland. The plant was severely stunted and young, unfolding leaves from the terminal bud were curled under and puckered, as shown by the photograph taken August 15 (Fig. 1). Careful examination of the nursery revealed only this one plant with curly top symptoms located about the center of the 2-acre nursery. Although there were approximately 200 different tobacco varieties in the field, the disease was on a plant of the Catterton variety commonly grown for leaf production in Maryland.

FIGURE 1. Symptoms of curly-top virus on Maryland tobacco at Plant Industry Station, Beltsville.

Three diseased plants of Coker 187-Hicks tobacco, similar in appearance to the plant in Figure 1 were observed in a 5-acre field at Oxford, North Carolina. These plants were confined to an area of about 1/20 acre near the center of the field. The presence of the curly-top virus in the affected plants from Beltsville, Maryland and Oxford, North Carolina was confirmed by leafhopper-transmission studies conducted by James E. Duffus, pathologist, Crops Research Division, Salinas, California. Successful inoculations were made to susceptible sugar-beet seedlings with leafhoppers which had fed on diseased tobacco tissue from plants at both locations. To confirm further the presence of curly-top virus in the original tobacco plants, the virus was transmitted from the infected sugar beets to healthy sugar-beet and tobacco plants. The effects on sugar beets indicate that the isolates were a relatively severe strain of the virus.

This is the first report of curly top on tobacco in Maryland and North Carolina. Curly top on tobacco had been observed in Kentucky in 1953 by Valleau et al. (12), in Wisconsin in 1954 by Fulton (2), and in Virginia in 1958 by Troutman and Fenne (11). In August 1958, Schneider (7) found curly top on sugar beets for the first time in Maryland. The sugar-beet field was on another farm at the Plant Industry Station about 4 miles from the tobacco field. Seemingly these occurrences of the disease were independent.

In 1934 at Shafter, California, which is out of the area of tobacco-leaf production of the United States, McMurtrey et al. (4) reported curly top in plantings of Nicotiana rustica and N. tabacum. The plantings were made to study nicotine production. At Shafter approximately 10 percent of the tobacco plants were affected with curly top, whereas consistently only one or two plants per acre have been found to date in fields in Wisconsin, Kentucky, Virginia, Maryland, and North Carolina.

In 1951, Morgan and McKinney (6) and later Morgan (5) described a disease similar in many respects to curly top but suggested it might be tobacco leaf curl as reported from Venezuela (13). Although Troutman and Fenne (11) in a review of occurrence of curly top state that Morgan and McKinney have reported the disease from Maryland, there are indications that it was neither curly top nor the tobacco leaf curl of Venezuela. The Maryland disease occurrence (5, 6) reported by Morgan and McKinney involved several hundred plants in two fields on the same farm. In all other reports of curly top on tobacco except the planting in California only one or two plants per acre or field were found. According to Morgan and McKinney (6) the virus particles were round, disk-like, and 75-80 mμ in diameter. The tobacco leaf curl of Venezuela (8) yielded virus particles approximately round but only about 39 mμ in diameter. Curly-top virus particles have been described (10) as rods, 20-30 x 150-200 mμ in size. Symptoms of the disease described by Morgan (5) have some similarity to those of a virus disease designated as curly dwarf and described by Slagg (9) as occurring in 1922 near Windsor Locks, Connecticut. Up to 20 percent of the plants in some fields were diseased. The same disease was observed in 1925 at Harrow, Ontario and at Albion Prairie, Wisconsin.

The plant affected with curly top at Beltsville (Fig. 1) showed evidence of recovery by mid-September. It was moved to an isolated place in the greenhouse, where it flowered and was judged normal in seed production. Seeds from the virus-infected plant were seeded in the Beltsville greenhouse during the winter, and 100 potted plants were held until blooming. Most plants were kept in a warm greenhouse at 75° to 85°F; however, some plants were kept for several weeks in a growth room at 62° to 70°. There was no evidence of seed transmission of the virus.

When the diseased plant was moved from the field to the greenhouse, root examination revealed a poorly developed secondary root system. Apparently, the growth of feeder roots as well as the top had been checked. Lackey (3) reported that some strains of curly top injure or kill the tiny feeder roots of beets.

The records of occurrences of curly top in 1958 on tobacco and sugar beets in Maryland and on tobacco in Virginia and North Carolina suggest a widespread occurrence of the vector. The viruliferous beet leafhoppers (Circulifer tenellus (Baker)) may have been blown into these States from a breeding area perhaps hundreds of miles to the west or south, as discussed by Douglass (1), who reported that abundant beet leafhoppers have been collected east of the Mississippi in Illinois and Florida. The diseased tobacco plants in Maryland and North Carolina were discovered about August 1, 1958. Only about one or two diseased plants per acre were found in fields with the disease. There may have been just a single period with winds and other conditions favorable for long-distance spread of the vector. Symptom expression on tobacco in Maryland, North Carolina, and Virginia (11) was very similar in August when diseased plants were found, whereas by mid-September evidence of recovery from acute symptoms was noted. These factors seem to suggest that infection occurred about the same time and that conditions were not favorable for secondary spread of the disease in the different tobacco fields.

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A VIRUS FROM FLORISTS' CHRYSANTHEMUMS WITH SOME CHARACTERISTICS OF TOBACCO MOSAIC VIRUS

Philip Brierley and Paul Lorentz[1]

Summary

A virus from chrysanthemum produced symptoms like those of tobacco mosaic virus (TMV) in Nicotiana glutinosa and Chenopodium amaranticolor, but failed to infect three varieties of beans (Phaseolus vulgaris). Symptoms differ slightly from those of TMV in Datura stramonium, tobacco and tomato. The virus is inactivated at 85° to 90° C, withstands dilution 10^{-6} and aging for more than 4 months at 20° C. It is not soil-borne. It appears poorly adapted to chrysanthemum.

In September 1957 C. J. Olson of Yoder Bros., Inc., Ashtabula, Ohio submitted tobacco plants infected with a TMV-like virus together with other viruses isolated in routine indexing of commercial chrysanthemums. The TMV-like virus produced local lesions of the TMV type in Nicotiana glutinosa and was discarded as a suspected contaminant. A soil-borne virus and a strain of cucumber mosaic virus also present in this material were reported earlier (1).

In November 1958 Olson supplied dry tobacco leaves showing intricate white etch patterns but little mottling, and also three florists' chrysanthemums that had repeatedly yielded the causal virus. Mechanical inoculation to tobacco yielded the same symptoms from the dry tobacco leaves and from the three chrysanthemum varieties. Some characteristics of the virus involved are reported herein.

SOME HOST REACTIONS

Turkish tobacco var. Samsun regularly developed white etch in addition to the green mottling characteristic of TMV, but never duplicated the intricate white etch pattern present in the original dried leaves. The virus isolated from the three chrysanthemum source varieties agreed with that from tobacco. N. glutinosa expressed necrotic local lesions indistinguishable from TMV local lesions. Datura stramonium developed local lesions, from which the virus often spread along a petiole to form a necrotic streak in the stem. Marglobe tomato developed inconspicuous black local lesions followed by systemic green mottling like that caused by TMV. Snapdragon expressed no symptoms but the virus was reisolated. Chenopodium amaranticolor produced yellow spots that later appeared systemically also; TMV induced the same sequence of symptoms in this plant. Three varieties of bean (Phaseolus vulgaris), namely Bountiful, Pinto and Refugee, developed no lesions in response to inoculation with the chrysanthemum virus, but all expressed the usual local lesions when inoculated with TMV.

Chrysanthemum Reactions

Olson reported that the three source varieties, two of domestic and one of European origin, yielded the virus in routine indexing on tobacco in July and October 1958. We recovered it from these plants in November and in January 1959, at which time the titer was low in young leaves. However, propagations made February 18 from these large source plants yielded no virus in March or April. Young, mature, and older leaves all tested negative on N. glutinosa. Evidently the virus was lost from these chrysanthemums in the usual practice of propagation by cuttings. Large plants of the original source varieties were flowered in April 1959; no defects attributable to the virus were detected in leaves or flowers.

The varieties Good News and Fred Shoesmith were mechanically inoculated with the virus from tobacco in December 1958. Six weeks later the virus was recovered in N. glutinosa from

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the inoculated leaves of both varieties and from leaves immediately above those in the variety Fred Shoesmith. It could not be recovered from upper leaves of either variety in January, February, March or April. We concluded that these varieties were not systemically invaded.

PHYSICAL PROPERTIES

Some physical properties of the chrysanthemum virus are compared with a TMV virus in Table 1.

Table 1. Some properties of the chrysanthemum virus and a comparison with a TMV type (Johnson from S. P. Doolittle).

Virus and date	Source	Test plants	Heating for 10 minutes (° C) 80°	85°	90°	95°	Dilution	Aging
Chrysanthemum virus:								
Dec. 12, 1958	Tobacco	Tobacco	3/3		0/3		10^{-6} +	>25 days
Jan. 30, 1959	do.	N. glutinosa	+[a]	+	-	-	10^{-6} +	> 4 months
Apr. 28, 1959	do.	do.	+	+	-	-	10^{-6} +	--[b]
Tobacco Mosaic virus:								
Apr. 28, 1959	do.	do.	+	+	+	-	10^{-6} +	--

[a] + = test positive, - = test negative.

[b] Untested.

A TEST OF SOIL TRANSMISSION

Because the symptoms in tobacco caused by the TMV-like virus from chrysanthemum resembled some of those associated with the soil-borne viruses described earlier (1), a test of soil transmission was made. Soil from pots in which infected tobacco had grown was freed from larger root residues, mixed and distributed to 72 thumb pots in which tiny tobacco seedlings were set. At the end of 10 weeks 5 of 72 plants showed green mottling in the tops. However, 10 tops without symptoms induced only 10 local lesions on 4 inoculated N. glutinosa, and 10 sets of roots produced no lesions on 4 plants. Evidently this virus, like TMV, persists for a time in root residues, but failure to reach a high titer in the roots of test seedlings shows that it is not typically soil borne.

DISCUSSION

There seems to be no record of TMV infection in chrysanthemum. Grant (2) and Holmes (3, 4) listed 27 species of Compositae susceptible to TMV, but none of these fall in the Anthemis tribe, which includes chrysanthemum. The chrysanthemum virus discussed here resembles TMV except for lower heat inactivation point and some differences in host range, such as failure to infect beans. Therefore we were inclined to consider it a distantly related strain of TMV. However Hugh Sisler, of the University of Maryland, found that the chrysanthemum virus fails to precipitate with TMV antiserum at any dilution whereas a known TMV tested in parallel reacted with this antiserum at dilutions up to 1:512.

Our failure to establish systemic infection with this virus in the chrysanthemum varieties Good News and Fred Shoesmith, together with the surprising experience of losing the virus from the original source chrysanthemums in the course of propagating these, indicates that this virus is poorly adapted to chrysanthemum. Perhaps it invades chrysanthemum from some other host in which it attains higher titer.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

CHLOROTIC SPOT, A GRAFT-TRANSMISSIBLE DISEASE OF LIGUSTRUM

A. G. Plakidas

In the summer of 1953, Mr. Edwin Lott, District Entomologist, New Orleans, Louisiana sent to this laboratory a specimen of waxleaf ligustrum (Ligustrum lucidum Ait.) leaves with numerous yellow spots (Fig. 1) for diagnosis, inquiring specifically if this disease were caused by a virus. He stated further that he had seen two ligustrum bushes in two separate nurseries showing this striking condition. The disease was unknown to the writer. Sometime later, one of the two sick bushes (about 3 feet in height) was taken to Baton Rouge and planted in the lath house for observation and study.

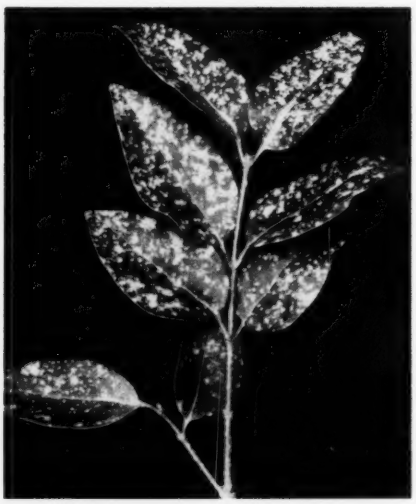

FIGURE 1. Chlorotic spotting as it appeared on the original, naturally infected Ligustrum lucidum bush.

DESCRIPTION

The symptoms of this disease are very striking. The foliage is covered with very numerous pale yellow to bright yellow spots (Fig. 1) varying in size from pin-head to about 5 mm, circular to irregular in shape. Discrete spots are mostly circular; larger chlorotic areas which result from the coalescing of several small spots are irregular in shape. Some of the discrete lesions are in the form of a chlorotic ring with minute green dot in the center. A few of the lesions are necrotic, reddish brown in color, especially on the older leaves. The chlorotic areas are depressed, thus giving the surface of the leaf a pock-marked appearance. The earliest symptoms appear on the new growth in the spring. Some, but not all, of the new leaves on each new shoot show large, irregular, diffuse, pale green to yellow blotches and puckering and malformation of the leaf blade (Fig. 2).

FIGURE 2. Diffuse yellow splotching, puckering, and malformation of young leaves.

TRANSMISSION

No fungus or bacterial pathogen was found associated with the disease, and all attempts to isolate a pathogen from the affected tissues by the usual laboratory techniques were unsuccessful. This suggested that the disease might be either a genetic abnormality or virus-induced. The grafting experiments reported below resulted in transmission of the disease to healthy plants, indicating that the disease is of virus nature.

Two graft-transmissible diseases of Ligustrum spp. have been described. Baur (1) described an infectious chlorosis of L. vulgare in Germany, and Hildebrand (2) a ring spot of privet (L. ovalifolium) in Texas. The symptoms of neither of these fit those of the present disease.

In November 1954 two L. lucidum plants growing in large (12-inch) pots were approach-grafted to branches of the original diseased bush. First symptoms, diffuse chlorotic splotches, appeared on the new growth of the grafted plants in the spring of 1955, and chlorotic spots on the mature foliage by the end of June of the same year. The symptoms were similar to but milder than those on the original bush. The spots were smaller, fainter, and less depressed than on the original bush (Fig. 3).

FIGURE 3. Symptoms on foliage of healthy plant approach-grafted to the original diseased bush.

In a second experiment, two healthy L. lucidum plants growing in 12-inch pots were cut back and cleft-grafted with scions from the original diseased bush and from healthy plants. Infection of the growth of the healthy scions occurred in both cases, but there were differences in symptom expression in the two plants. In one plant, the symptoms were similar to those resulting from the bridge-grafts. In the second plant, the large, diffuse, chlorotic splotches that developed on the young leaves persisted on the older, mature leaves, but the smaller, definite chlorotic spots were absent.

In a third experiment, scions were taken from five different healthy bushes picked at random on the University campus and cleft-grafted separately on five shoots of the original diseased bush. The object of this test was to determine whether there are any differences in susceptibility in different clones. Only three of the five grafts took and grew. Differences in symptom expression were noted. One of the three successful grafts developed typical symptoms that approached those of the original bush in severity; the symptoms on the second graft were similar but milder, fewer and fainter chlorotic spots. The third graft showed larger diffuse pale-green splotching and puckering of the young foliage but no small chlorotic spots on the mature leaves.

No effort was made to transmit the disease by any method other than grafting. No juice inoculations were made, and no attempt was made to find possible insect vectors.

Later observations showed that what appears to be the same disease, but in a mild form, is fairly common in the Baton Rouge area. Many waxleaf ligustrum shrubs and hedges on the University campus, as well as in private yards, were noted showing yellow splotching, puckering, and malformation of young foliage in the spring and a scattering of faint chlorotic spots on the mature foliage. These symptoms are mild enough to be easily overlooked. This species of Ligustrum is commonly propagated vegetatively from cuttings. However, it also grows very readily from seed, and large numbers of volunteer seedlings often occur on vacant ground in the vicinity of large bushes. Many of these volunteer seedlings are utilized by nurserymen and home owners for landscaping. Variability among seedlings, including variability in susceptibility to disease, would be expected. On the basis of observation and of admittedly limited experimental data, it is believed safe to postulate that: 1) this graft-transmissible disease, presumably of virus nature, is relatively widespread among ligustrums, 2) the great majority of the plants are tolerant of the virus and exhibit only mild symptoms that are easily overlooked, and 3) a few nontolerant plants exhibit the strikingly severe symptoms described in this paper. The existence of an insect vector is also postulated.

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DATURA STRAMONIUM L., A POSSIBLE QUANTITATIVE BIOASSAY HOST FOR THE BROMEGRASS MOSAIC VIRUS[1]

Ren-jong Chiu and Webster H. Sill, Jr.[2]

Summary

When Datura stramonium L. was manually inoculated with a collection of bromegrass mosaic virus (BMV), local chlorotic rings with a light green center were produced on the plant. Systemic infection was observed in plants from which apical shoots were removed immediately before inoculation. Local response to the same virus, expressed as faint chlorotic solid spots or lesions, was also produced in Chenopodium album L. Since D. stramonium possessed larger leaves and produced discrete, easily countable lesions, it was chosen over C. album as a possible quantitative assay host. These two plants were the only ones that developed symptoms among 33 species or varieties tested.

The number of lesions produced on leaves of D. stramonium was directly related to the concentration of virus in diseased leaf sap when the latter was diluted beyond 10^{-2}. Two types of lesions, which may indicate two strains of BMV, were produced by the 15 collections of BMV tested. Fourteen collections produced lesions as described above, but one, the type culture, had a longer incubation period and produced only occasional faint chlorotic lesions which often gradually became rather large. The virus was reisolated from these plants, and on other diagnostic hosts (Bromus inermis Leyss., Golden Giant sweet corn, wheat, and barley) symptoms from all collections appeared to be similar.

McKinney (2, 3) reported local increase of the bromegrass mosaic virus (BMV) and lesion formation in several dicotyledonous plants, including garden bean var. Scotia, cucumber var. Early White Spine, tobacco var. Samsun, garden beets, sugar beets, and swiss chard. However, these plants, when tested, were not considered suitable for quantitative assay purposes and several gave no response to the virus in this study. An urgent need for a satisfactory local lesion bioassay host for this virus prompted the authors to make a continuing extensive search, the results of which are reported here.

MATERIALS AND METHODS

All plants tested for reaction to BMV inoculation were pot-grown in a greenhouse maintained at a temperature around 75° F. A single BMV collection originating from naturally infected smooth bromegrass, Bromus inermis Leyss., and capable of causing top necrosis in Golden Giant sweet corn (3, 4, 6) was used in most trials. Other known BMV collections were used only for confirmative and comparative tests when necessary. With eight collections the virus was increased in young wheat or barley plants but with seven others it was obtained from chopped and desiccated diseased wheat or B. inermis leaves. To prepare inoculum the leaves were ground with mortar and pestle, diluted as desired with tap water, and squeezed through several layers of cheesecloth. Plants were inoculated where possible during the seedling stage by the common carborundum leaf rubbing technic, either with the fingers or with cotton swabs. To control mites and insects, a weekly greenhouse fumigation was conducted with Plantfume 103 (a commercial smoke generator containing 15 percent tetraethyl dithiopyrophosphate as the active ingredient).

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EXPERIMENTAL RESULTS

Varieties within 21 species of plants in seven families were tested for their response to mechanical inoculation with BMV. A complete list of those plants is given in Table 1.

Table 1. Reactions of different plant species or varieties to mechanical inoculation with bromegrass mosaic virus.

Plant name	Reaction
Cucurbitaceae:	
Cucumis sativus L. var. Chicago Pickling	No symptoms
White Spine	do.
Ohio MR 17	do.
Palmetto	do.
C. melo L. var. Rockyford	do.
Hale's Best	do.
Cucurbita pepo L. var. Connecticut Field	do.
C. maxima Duchesne var. Hubbard	do.
Bush Scallop	do.
Leguminosae:	
Phaseolus vulgaris L. var. Tender Green	do.
Scotia	do.
Pinto Field Bean	do.
Idaho Refugee Bush	do.
Refugee Green Bush	do.
Kansas Wonderful	do.
P. lunatus L.	do.
Vigna sinensis Savi. var. Black Eye	do.
Vicia faba L. var. Broad Wonderful	do.
Pisum sativum L. var. Alaska	do.
Solanaceae:	
Capsicum frutescens L.	do.
Nicotiana glutinosa L.	do.
N. rustica L.	do.
N. megalosiphon Heurck & Muell.	do.
N. tabacum L. var. Samsun	do.
Havana 38	do.
Datura stramonium L.	Chlorotic rings
D. meteloides Dunal	No symptoms
Amaranthaceae:	
Gomphrena globosa L.	do.
Scrophulariaceae:	
Antirrhinum majus L.	do.
Compositae:	
Zinnia elegans Jacq.	do.
Chenopodiaceae:	
Beta vulgaris L.	
Garden beet var. Early Wonder	do.
Swiss chard var. Lucullus	do.
Chenopodium album L.	Faint chlorotic spots on inoculated leaves

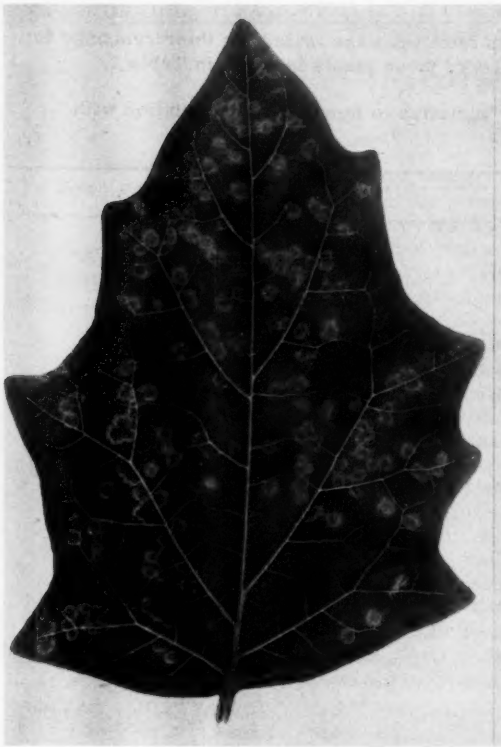

FIGURE 1. Local lesions induced by a BMV collection on inoculated leaf of Datura stramonium. Picture taken 10 days after inoculation.

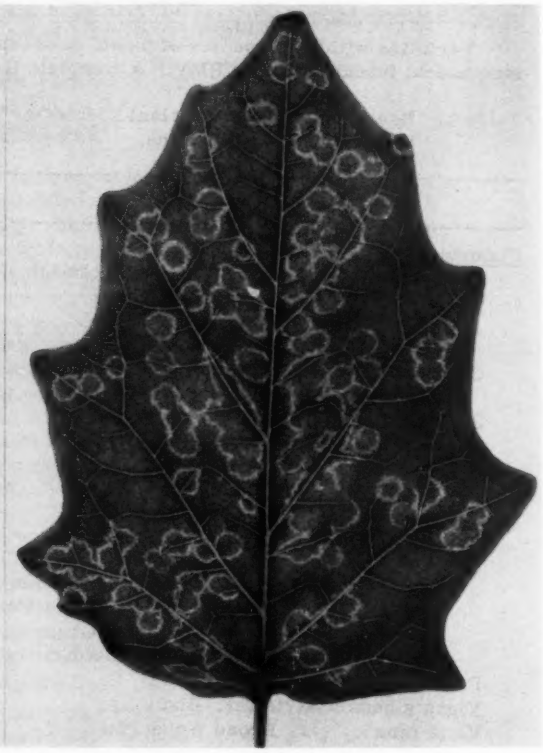

FIGURE 2. Unusually large lesions produced on inoculated leaf of Datura stramonium under summer greenhouse conditions. Picture taken 15 days after inoculation.

From 4 to 12 plants were inoculated for each test and at least 2 plants were similarly rubbed with water and carborundum to serve as controls. No test for the recovery of virus from inoculated plants was attempted unless symptoms appeared. Of those plants listed, only D. stramonium and C. album produced chlorotic rings and faint chlorotic spots, respectively. D. stramonium, possessing larger leaves and producing discrete, easily countable lesions, appeared to be better than C. album as a local lesion host for quantitative study. Later work was, therefore, confined to this species.

The presence of the virus in infected D. stramonium was proved by inoculation from it to several susceptible hosts. It caused top necrosis in Golden Giant sweet corn and typical systemic mottling and streaking in B. inermis, wheat, barley (3, 4, 6), and Poa pratensis L. (7) indistinguishable from symptoms produced by the original virus culture, as well as the type culture of BMV (5)[3]. A total of 14 recent Kansas collections of BMV, 13 of which were from smooth bromegrass and one from P. pratensis, have so far been inoculated to D. stramonium. All of these induced similar local lesions which appeared 4 to 6 days after inoculation as circular chlorotic ring spots, usually 1 to 2 mm in diameter, with normal green or light green tissue at the centers (Fig. 1). Lesions gradually enlarged to a limited extent and sometimes coalesced. On highly susceptible leaves, where numerous lesions were crowded together, lesions often took the form of solid chlorotic spots about pinhead size. This was typical of the response of the second or third true leaf when inoculated with fairly concentrated inoculum (10^{-1} to 10^{-2}). Unusually large lesions with a diameter of around 10 mm (Fig. 2) have also

[3]Collection No. AC-66, received from American Type Culture Collection, 2029 M Street, N. W., Washington, D. C.

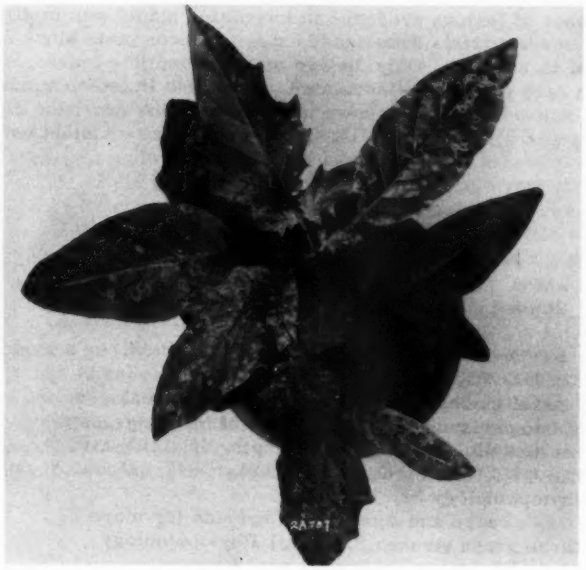

FIGURE 3. Systemic symptoms which developed on Datura stramonium after removal of apical meristems, consisting of discrete chlorotic spots, rings and oak-leaf patterns which developed in noninoculated leaves arising from lateral buds. Primary local lesions on two lowest inoculated leaves can also be seen. Picture taken 28 days after inoculation.

been observed under spring and summer greenhouse conditions.

The type culture of BMV (5) had a longer incubation period on D. stramonium and caused only occasional faint, chlorotic spots which gradually became quite large (8 to 18 mm in diameter). However, when reisolated from D. stramonium, all BMV collections tested, including the type culture, behaved essentially alike in the diagnostic hosts Golden Giant sweet corn, B. inermis, wheat, and barley seedlings (3, 4, 6). Hence, the type culture of BMV and all other collections studied are probably the same virus. The two types of local lesions developing in D. stramonium may well indicate the existence of two different strains. Lal and Sill (1) presented other evidence for the existence of at least two strains of BMV in a recent study of synergistic reactions in small grain virus combinations.

Table 2. The relation of the dilution of BMV to the number of local lesions produced on Datura stramonium.

	Number of lesions[a] produced by BMV when taken from		
	Barley		Poa pratensis
Dilution	Test 1	Test 2	
$10^{-1.0}$	88.0	232.3	289.5
$10^{-1.5}$	81.5	244.5	286.9
$10^{-2.0}$	78.8	235.5	228.8
$10^{-2.5}$	47.0	133.8	157.9
$10^{-3.0}$	12.4	52.1	76.6
$10^{-3.5}$	3.1	9.0	23.0
$10^{-4.0}$	0.4	3.5	3.3
$10^{-4.5}$	0.4	0.5	0.1

[a]The number of lesions is an average of 16 half-leaves in tests with barley and of 8 half-leaves in the test with P. pratensis.

The removal of young shoot and flower buds before inoculation often resulted in systemic spread of the virus in D. stramonium. Young leaves rising from the lateral buds of such inoculated plants developed chlorotic spots or chlorotic rings. The rings, if across a leaf vein,

often elongated along the vein to assume a somewhat oak-leaf pattern (Fig. 3).

To determine whether the number of lesions produced in inoculated plants was in direct proportion to virus concentration, several trials were made using the incomplete block design (9) in combination with the half-leaf technic (8). Only the two most susceptible leaves, usually the third and fourth true leaves, in each plant were inoculated. Decrease in lesion numbers resulted from a decrease in virus concentration, as shown in Table 2. This decrease did not become apparent until the inoculum was diluted beyond 10^{-2}, but was quite consistent between $10^{-2.5}$ and 10^{-4}.

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SPREAD OF VIRUS INTO STRAWBERRY NURSERY STOCKS

John R. McGrew[1]

Abstract

Indexing to the East Malling clone of Fragaria vesca of 140 plants sampled from 12 varieties of strawberry grown in the field for 2 to 6 years on the Eastern Shore of Maryland and in Delaware by three commercial nurseries revealed that six plants had become infected with mild mottle virus. All infected plants were of two varieties from a single nursery.

Posnette and Bovey (4) reported that 0, 10, 50, and 100 percent of virus-free strawberry plants sampled became infected in 6 months, 1, 2, and 3 years respectively. Their work was carried out at East Malling, England, with limited isolation from other virus sources (only 1-200 yards), but with aphid control.

The efficiency of control of virus movement into strawberries grown commercially under conditions of extensive isolation (1000 yards) and of aphid control is reported in this paper.

MATERIAL AND METHODS

Stocks of several strawberry selections and varieties free of viruses detectable in the East Malling clone of Fragaria vesca (EMC) were supplied by the United States Department of Agriculture to three nurseries under a cooperative agreement, beginning in the spring of 1951. The agreement required that the plants be grown in fields isolated by at least 1000 yards from other plantings of strawberries and from wild strawberries to reduce the chance of aphids bringing viruses into the plantings. In addition, the plantings were to be dusted at biweekly intervals with parathion from early spring to late fall to prevent possible secondary spread of viruses by aphids within the fields.

Two bundles of 25 plants each of 12 specified varieties were requested from each of the three nurseries in the spring of 1957. Plants supplied by the nurseries were from the same stocks used to fill customer orders.

Two plants were taken at random from each bundle. The selected plants of some varieties were injured in storage and were weak or failed to grow. These were replaced with additional plants from the same bundles. In some cases more than four plants of a variety were finally indexed.

When runners formed, plants were indexed to EMC. All graft unions were checked by Goheen's method (3). Plants which failed to runner were indexed to EMC by the excised leaf technique (1). Three leaflets were grafted and plants were regrafted unless at least two leaflets remained green and turgid for 5 weeks or symptoms appeared in the EMC. In addition, one runner plant of the excised leaflet-grafted EMC was allowed to root in a 3-inch pot so that there would be rapid growth that would better express virus symptoms.

RESULTS

Results of the index tests are given in Table 1. The exact number of years the indexed plants of 8 of the 12 varieties were grown in the field was impossible to determine as stocks of these had been supplied to the nurseries more than 1 year.

All plants sampled from two of the nurseries were free of detectable viruses. Some, but not all, plants of two varieties from the third nursery had become infected with strawberry mild mottle virus. The symptoms produced in the EMC indicator plants varied. The variation indicates infection by different strains of mottle virus (2) rather than sampling several plants from single infected clones.

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Table 1. Sample indexing for virus infection of strawberry plants grown for 2 to 6 years in three nurseries on the Eastern Shore of Maryland and in Delaware under conditions of 1000-yard isolation and frequent dusting with parathion.

Variety[a]	Years in field[b]	Number of plants					
		Nursery A		Nursery B		Nursery C	
		Non-infected	infected	Non-infected	infected	Non-infected	infected
Armore	3-4	4	0	4	0	4	0
Blakemore	3-5	4	0	4	0	4	0
Catskill	3-5	3	0	6	0	4	0
Dixieland (s)	6	3	0	3	0	6	0
Florida Ninety	2	4	0	6	0	4	0
Gem	2	-	-	4	0	4	0
Howard 17	3-5	2	0	4	0	5	0
Pocahontas (s)	6	3	0	4	0	4	0
Redglow (s)	2-3	1	3	3	0	5	0
Sparkle	3-5	-	-	4	0	4	0
Surecrop (s)	2-3	2	3	4	0	5	0
Tennessee Beauty	3-6	4	0	5	0	4	0

[a](s) indicates supplied by U. S. Department of Agriculture before date of introduction.
[b]Determined from first and last year supplied to nurseries.

DISCUSSION

The limited sampling does not assure that the stocks which showed no virus had remained 100 percent virus-free, but under the conditions on the Eastern Shore of Maryland and in Delaware, 1000-yard isolation from sources of strawberry viruses plus frequent aphicide dusting appear to give a practical and satisfactory protection of strawberry nursery stock against virus infection.

It was not possible to determine whether the mottle virus in the two infected stocks resulted from insufficient isolation from outside sources of viruses or from a failure to control secondary spread within the planting from an occasional virus-infected plant.

Because control of virus in two instances was less than desirable the advisability of replacing field stocks with new virus-free foundation stock every 2 or 3 years is emphasized.

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THE EFFECT OF ALFALFA MOSAIC VIRUS ON ROOT DEVELOPMENT OF ALFALFA CUTTINGS[1]

J. D. Panzer[2]

While clonal lines of alfalfa from cuttings were being established, certain cuttings seemed to grow poorly in the cutting bench. It was also noted that certain of the cuttings were infected with the alfalfa mosaic virus. To test the possibility that the poor root development was the result of virus infection the following experiments were conducted.

A one-half-year-old vernal alfalfa plant was cut in two so that separate clonal plants were obtained. One of these plants was inoculated mechanically with alfalfa mosaic virus. After the disease had developed, cuttings were made from both the infected and the non-infected plant. The cuttings were allowed to root, after which they were planted in sterile soil in the greenhouse (winter conditions). In this way 25 diseased and 25 non-diseased clonal alfalfa plants were established. Diseased plants were tested for virus by local lesion assay on the common bean Phaseolus vulgaris var. Bountiful.

RESULTS

In the first experiment 45 cuttings, approximately 1 inch long, were made from the diseased clonal plants. In a like manner, 36 were made from the healthy plants. After a majority of the leaves were removed, the cuttings were placed in a cutting bench filled with Perlite and automatically misted with water once each minute to prevent desiccation. At the end of 37 days the cuttings were removed, the length of the root system measured, and the number of roots developing from the crown determined.

The average root length was 3.7 cm for the diseased as compared with 6.6 cm for the healthy plants. This was a reduction of 44 percent, which was significant at the 1 percent level ($T = 4.9$; $T_{01} = 1.99$).

The average number of roots was 3.7 for the diseased plants and 5.4 for the healthy ones. This reduction of 32 percent was significant at the 1 percent level ($T = 5.7$; $T_{01} = 1.99$).

A second similar experiment was made later when growing conditions in the greenhouse were more favorable due to longer days, higher light intensities, and higher temperatures. In this experiment 39 virus infected and 37 healthy clonal cuttings were used.

The average root length for the diseased plants was 4.9 cm as compared with 6.2 cm for the healthy plants. The 21 percent reduction in root length was significant at the 1 percent level ($T = 3.6$; $T_{01} = 2.6$).

No difference was noted in the number of primary roots developing from the crown; however, secondary root development was more advanced in the healthy than in the virus infected plants. Total root weight (fresh weight) for the diseased plants was 4.9 grams and 6.2 for the healthy ones.

DISCUSSION AND SUMMARY

The alfalfa mosaic virus seems to affect clonal propagation of alfalfa by impairing root development and growth. This impairment appears worse during poor growing conditions. Since the establishment of plants from cuttings is reduced by plants with poor root development and since virus-diseased plants are usually not desired, it is suggested that "poor rooter" clones be tested for virus infection.

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A RECENTLY DISCOVERED VIRUS-INDUCED STREAK OF PEAS

W. J. Zaumeyer[1] and Graciano Patino[2]

Summary

A new streak virus isolated from peas in southern Idaho is described, identified, and compared with other pea streak viruses. The symptoms produced by the new virus are similar to those of four previously reported pea streak diseases and a closely related virus of sweetclover reported from Germany. The most conspicuous symptoms of the new disease are a purple necrotic streaking of the stems and a necrotic spotting, pitting, and malformation of the pods. In the greenhouse the symptoms are not nearly so striking as they are in the field. All 119 pea varieties and strains tested were susceptible. Among 43 plant species representing eight families tested, seven, representing two families, were found susceptible. The virus was inactivated at 65° to 70° C in 10 minutes and by 4 days in storage in vitro at about 22°. It withstood dilution of 1:5,000, but not 1:10,000. On the basis of host range and physical properties, the virus is regarded as a new strain of the pea streak virus.

In a survey for pea diseases in the Pacific Northwest in 1956 a virus-induced streak of pea, Pisum sativum L., was found in many fields in southern Idaho (11). It caused considerable yield reduction in areas where it was widespread. In 1957 it was reported to be the most serious virus disease of peas ever noted in the area. What was possibly the same disease was noted by the senior author in Idaho in 1932 and 1934 and in Montana in 1934 (10). No virus determination was possible at that time, but the nature of the disease and its symptoms were similar to those reported here. The symptoms of the new disease resemble those of virus-induced pea streaks previously described by Zaumeyer (10), Hagedorn and Walker (2), and Kim and Hagedorn (3) in the United States, and by Chamberlain (1) in New Zealand. They also resemble those produced by the white sweetclover virus reported by Quantz and Brandes (6) in Germany. Serological investigations by Wetter and Quantz (8) indicated that this last virus is related to the Wisconsin pea streak virus and to the I-5 isolate of an Idaho streak virus (3).

Plants naturally infected with the virus collected in Idaho by the senior author were sent to Beltsville, Maryland to determine the possible relationship between this virus and the other described pea streak viruses. This paper reports the results of this study.

MATERIALS AND METHODS

The greenhouses in which all the studies were conducted were maintained at approximately 22° to 28° C. Pea streak-infected plants sent from Idaho were desiccated and so stored for about 4 months under refrigeration before inoculations were made to peas and other species. During the course of the study the virus was maintained on plants of several varieties of peas and broadbean. Carborundum powder No. 320 was used as the abrasive in the transfers. The plants were inoculated in the early stages of growth by wiping the leaves with a cheesecloth pad dipped in the inoculum.

SYMPTOMS ON PEAS

Under field conditions the most conspicuous symptoms on peas are purple to brown necrotic streaking of the stems and petioles (Fig. 1, D and H) and a necrotic spotting and malformation of the pods (Fig. 1, F). Stem and petiole streaking are usually noted first near the upper portion of the plant, but later stem streaking may extend from the top to the bottom.

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Pods forming when infection takes place do not develop seeds, remain flat and take on a dark-purplish-gray color (Fig. 1, F) over a considerable portion of their surface. They may also be spotted, pitted and distorted (Fig. 1, F). Pods with fully formed seeds develop only the purple pitted markings. The initial symptom is a slight purple streaking of the stems and lower portion of the stipules. The latter recurve (Fig. 1, H) and the leaflets curl downward. The veins of the leaves and stipules are often cleared. The stem internodes of the upper part of the plant are shortened, and the leaves, which are smaller than normal, are rosetted and slightly chlorotic but not mottled. The apical growing points usually curl to one side, and the stems and petioles become very brittle. The veins and veinlets of the leaves and stipules may become necrotic, and often necrosis is observed in the interveinal tissues. Infected leaves finally become flaccid, the tip of the plants may wilt and often whole plants die (Fig. 1, D). In many cases, however, only the terminal growing points die. Some of these symptoms are not found on all infected plants, and many variations are noted if only some symptoms appear.

The symptoms are not nearly so striking in the greenhouse as in the field. Although the first evidence of the disease is a reddish-brown necrotic streaking of the stems and petioles, the most characteristic symptom is wilting of the plants (Fig. 1, D). The stem discoloration is most conspicuous at the nodes. Small necrotic spots, slight vein necrosis, and vein clearing (Fig. 1, C) are often noted on the leaves and stipules above the inoculated ones. These leaves wilt and die, and progressive wilting and death of the upper leaves and stipules follow. Often seedlings die about 15 days after inoculation.

INOCULATION OF PEA VARIETIES

Since no resistant varieties of peas were found in the survey in southern Idaho in 1956, a large number from various commercial sources were tested later. It was hoped that several would be found that showed resistance so that a breeding program could be initiated. One hundred and nineteen varieties and strains were inoculated with the streak virus, and all were found susceptible (Table 1).

Five varieties (Yellow Admiral, Eureka, Nome, Pedigree Extra Early, and Willet Wonder) showed mild symptoms, 10 moderate, 94 severe, and 10 were killed by the virus (Table 1). A high percentage of the inoculated plants of most varieties became infected. However, in 23 varieties a relatively high percentage of healthy plants were noted. Of the 23, 3 were classified as mildly infected, 3 as moderately, and 17 as severely infected. The uninfected plants were not reinoculated to determine definitely whether they were resistant or had escaped infection. In many cases, however, two separate inoculation tests were made and uninfected plants were noted in each test (Table 1).

INOCULATION OF OTHER SPECIES

Besides peas, 43 species representing 8 families were inoculated. Eight species, of which seven were from the family Leguminosae, were susceptible. Responses of the susceptible species were as follows:

Cicer arietinum L. (chickpea). Leaf necrosis and killing of terminal leaflets followed by death of plant.

Glycine max (L.) Merr. (soybean) vars. Jackson, Ogen and Roanoke. Yellow stipple-like mottle; green vein banding (Fig. 1, A).

Gomphrena globosa L. (globe amaranth). Reddish necrotic local lesions.

Melilotus alba Desr. (white sweetclover). Mild mottle.

Lens culinaris Medik (lentil). Stem discoloration, wilting and death.

Trifolium subterraneum L. (subclover). Mild mottle.

Vicia villosa Roth. (hairy vetch). Reddish brown local lesions; necrotic stem lesions.

Vicia faba L. vars. minor Peterm. (bell bean) and major Harz (broad windsor). Dark-brown primary necrotic lesions (Fig. 1, B); systemic leaf necrosis (Fig. 1, E); stem streaking (Fig. 1, G); and death of plants.

No infection was obtained in Antirrhinum majus L. (snapdragon), Astragalus cicer L. (cicer-milk vetch), Brassica oleracea var. botrytis L. (broccoli), Cajanus cajan (L.) Druce (pigeon pea), Canavalia ensiformis (L.) DC. (jackbean), Capsicum frutescens L. (pepper), Crotalaria spectabilis Roth (crotalaria), Cucumis sativus L. (cucumber), Cyamopsis tetragonoloba (L.) Taub. (guar), Datura stramonium L. (jimsonweed), Lupinus albus L. (white lupine), L. angustifolius L., Medicago sativa L. (alfalfa), Nicotiana glutinosa L., N. rustica L., N. sylvestris Speg., N. tabacum L. (tobacco var. Samsun), Petunia hybrida Vilm.

Table 1. Susceptibility of pea varieties to the Idaho pea streak virus.

Variety	Plants (number) Inoculated	Infected	Symptoms[a]
Ace	17	17	D
Admiral, Green	17	17	C
Admiral, Yellow	53	20	A[b]
Advancer	16	16	C
Alaska	12	10	C
Alaska 2 B	19	17	C
Alaska, Rogers 123	13	10	C
Alaska, Rogers 423	18	17	C
Alaska, Wilt Resistant	12	12	D
Alderman	17	17	C
Alderman Early	16	16	C
Apex	22	22	D
Banqueteer	17	15	C
Black Eye Marrowfat	13	12	B
Bonneville	11	11	C+
Bountiful	25	24	C
Canner Gem	11	11	C
Canner Hybrid	10	10	C
Canner Hybrid No. 57391	11	11	C
Canner King	9	9	C
Carter Eight Weeks	16	15	C
Champion of England	12	12	C
Chief	15	14	C
Duke of Albany	22	21	C
Dwarf Defiance	17	16	C+
Dwarf Grey Sugar	20	17	C
Early Bird	16	14	C+
Early Harvest	21	16	C
Early Perfection	12	12	C
Early Sweet	13	9	B
Elf	11	4	C
Eureka	13	13	A
Extra Early	14	14	C
Famous	15	15	C
Freezer Hybrid	15	14	C
Freezer 33	14	13	C
Freezer 37	17	17	C
Frostee	12	11	C
Gallatin No. 2	17	17	D
Glacier	13	12	B
Gradus	13	13	C
Gradus, Improved	8	8	B
Hardy	11	11	C
Harvester Early	7	7	C
Horsford	16	13	C
Hundredfold	18	16	B
Imperial 58237	25	25	C
Jade	18	18	C
Laurel	15	12	C
Laxton No. 7	14	11	C
Laxton No. 8	15	13	C
Laxton Progress	18	17	D
Laxton Superb	21	20	C+
Lincoln	18	17	C
Little Marvel	27	25	C
Lolo	18	18	C+
Loyalty	16	15	C
Major	15	15	C
Mammoth Luscious Sugar	24	14	C
Mammoth Melting Sugar	23	14	B
Midfreezer	12	11	C
Miracle	17	15	C
Miragreen	16	16	D
Morse No. 60	13	13	C
Multifold	12	10	C
New Era	13	13	C+
No. 13	30	21	C
No. 40	8	7	C
No. 361	16	11	C
Nome	43	13	A[b]
Nott Excelsior	36	36	D[b]
Onward	27	12	C[b]
Pacemaker	17	17	D
Pedigree Extra Early	31	8	A[b]
Perfected Freezer	11	11	C+
Perfected Wales	15	15	C
Perfection	24	19	C
Perfection, Dark Seeded	17	17	D
Perfection, Early	18	18	D
Perfection (Rogers 194)	13	12	C+
Perfection, Wisconsin	15	15	C
Perfection Wilt Resistant	14	14	C+
Pilot	14	9	C
Pioneer	26	15	C
Pixie	33	19	C[b]
Pluperfect	21	7	B
Premium Gem	38	22	C[b]
President Wilson	9	9	B
Pride	19	11	C
Prince Edward	16	12	C
Prince of Wales	31	15	C[b]
Profusion	16	16	C
Profusion, Improved	9	9	B
Progress No. 9	21	13	C
Prolific Early Market	16	10	C
Rainer	14	14	C
Rondo	35	32	C[b]
Rice's Early Dwarf	8	5	C
Senator	16	10	C
Shasta	27	16	C
Shoshone	15	14	C
Signal	14	9	C
Small Seeded Freezer	21	21	C
Stratagem, Improved	8	8	C
Surpass	28	18	C[b]
Surprise	14	14	C+
Sutton Excelsior	17	16	C
Teton	26	7	C
Thomas Laxton	21	19	C+
Victory Freezer	10	10	C
Viking	15	15	C+
Wasatch	13	13	C+
White marrowfat	40	22	C[b]
Willet Wonder	23	19	A
Winner	18	15	C
World Record	30	26	C
Wyola	50	27	C
Yorkshire Hero	33	17	B[b]
Yukon	37	18	C

[a] A = mild; B = moderate; C = severe; D = dead.
[b] Two tests.

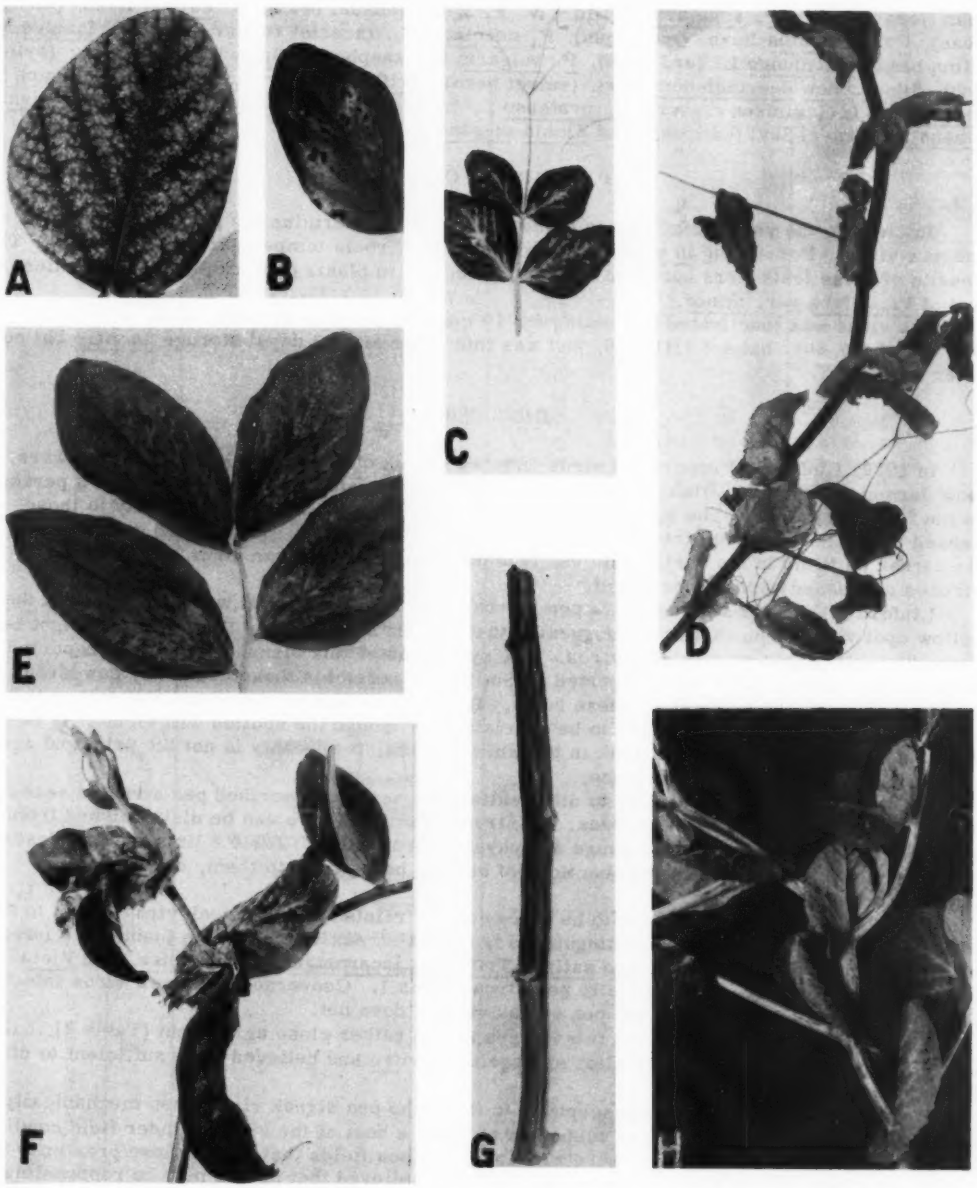

FIGURE 1. Symptoms produced by the Idaho pea streak virus on several hosts: A -- Mottle and green vein banding on Ogden soybean. B -- Primary necrotic lesions on inoculated leaflet of broadbean. C -- Veinclearing on Perfected Wales pea. D, H -- Stem streaking on Laxton Progress and Little Marvel pea, respectively. E, G -- Systemic leaf necrosis and stem streaking of broadbean. F -- Pod malformation of Prince of Wales pea.

(petunia), Phaseolus aconitifolius Jacq. (moth bean), P. acutifolius Gray var. latifolius Freeman (tepary bean), P. angularis (Willd.) W. F. Wight (adzuki bean), P. aureus Roxb. (mung bean), P. calcaratus Roxb. (rice bean), P. coccineus L. (scarlet runner bean), P. lunatus L. (lima bean), P. mungo L. (urd bean), P. vulgaris L. (snapbean), Spinacia oleracea L. (spinach), Stizolobium deeringianum Bort. (velvet bean), Trifolium hybridum L. (alsike clover), T. incarnatum L. (crimson clover), T. pratense L. (red clover), Vicia sativa L. (vetch), Vigna sinensis (Torner) Savi (cowpea), and Zinnia elegans Jacq. (zinnia).

PROPERTIES OF VIRUS

Juice from the young infected pea plants was used to determine the physical properties of the new virus. For ageing in vitro, juice was stored at room temperature (about 22° C). The results of these tests were based on systemic infection in plants of Dark Seeded Perfection pea and of Vicia faba var. minor.

The virus was inactivated by heating for 10 minutes between 65° and 70° C, withstood dilution of 1:5,000, but not 1:10,000, and was infectious after 3 days' storage in vitro but not after 4 days.

DISCUSSION

In 1929, Linford (4) reported a virus-induced streak of peas from Maryland, Delaware, New Jersey, Wisconsin, Utah, Idaho and Montana, where infection varied from 2 to 5 percent in the fields examined. The symptoms of the disease showed a marked similarity to those caused by the several pea streak viruses mentioned in this paper. As Linford did not study the streak he found in the various States, it is not known whether one virus or any of the viruses mentioned here was involved.

Linford (5) in 1932 showed that a pea streak virus found in Hawaii was identical with the yellow spot virus of pineapple and suggested that the pea streak in the United States might be caused by either this or a related virus. The symptoms of this disease on pea, as reported by Whipple (9), and on Vicia faba, reported by Smith (7), resemble those caused by pea streak virus 1 and by the Idaho virus on these hosts. Differences in host range and physical properties, however, show these viruses to be unrelated. Although the spotted wilt virus may be responsible for some of the pea streak in the United States, it probably is not the principal agent in the field occurrence of the disease.

Although it may be impossible to differentiate the several described pea streak diseases by the symptoms they produce on peas, the streak described here can be distinguished from the others by differences in host range and physical properties. Table 2 lists the five described pea streak viruses, shows the reactions of certain host plants to them, and gives their physical properties.

The new streak virus appears to be more closely related to pea streak virus 1 than to any of the other viruses. It can be distinguished from pea streak virus 1 by its inability to infect by mechanical inoculation Medicago sativa, Trifolium incarnatum, T. pratense, and Vicia sativa, all of which are susceptible to pea streak virus 1. Conversely, the new virus infects Glycine max and Vicia villosa, but pea streak virus 1 does not.

The physical properties of the two viruses are in rather close agreement (Table 2), but the differences in thermal inactivation and ageing in vitro are believed to be sufficient to differentiate them.

Even though alfalfa was not susceptible to the Idaho pea streak virus when mechanically inoculated in the greenhouse, it is suspected of being a host of the virus. Under field conditions Idaho pea streak is usually more widespread in pea fields that are in close proximity to alfalfa plantings than in those that are not, and it is believed that aphids may be responsible for transmitting the virus from alfalfa to peas. This was also the case with the pea streak virus found in 1932 and 1934 in Idaho and Montana. Infected pea plants were more commonly found growing close to alfalfa plants either where volunteer plants were observed in a pea field or where alfalfa was growing along the border of or in an adjacent field. No suspicious symptoms have been found in field-grown alfalfa plants to suggest the presence of the streak virus, nor has the streak virus been isolated from this species. Hagedorn and Walker (2) recovered the Wisconsin pea streak virus from a number of inoculated species which showed no symptoms of the disease, but alfalfa was found to be resistant.

Table 2. Comparison of host reactions and physical properties of the reported pea streak viruses.

Virus[b]	Host reaction[a]														Properties		
	Pisum sativum	Cucumis sativus	Glycine max	Lathyrus odoratus	Lupinus angustifolius	Medicago sativa	Melilotus alba	Phaseolus vulgaris	Trifolium hybridum	T. incarnatum	T. pratense	Vicia faba	V. sativa	V. villosa	Thermal inactivation (°C.)	Dilution	Ageing in vitro (days)
Pea streak virus 1 (10)	+	-	-	-		+	+	-		+	+	+	+	-	62-65	1:5,000	1-2
New Zealand pea streak (Pisum virus 3) (1)	+	+	+	+	+	-		+	+		+			+	78-80	1:1,000,000	44-44
Wisconsin pea streak (2)	+	-	+	+[c]	-	-	+	-	+	+[c]	+	-	-	+[c]	58-60	1:1,000,000	16-32
German sweetclover virus (6)	+		-			+	+	-	-	+[c]	-	+		+[c]	68-70	1:100,000	1-2
Idaho pea streak virus	+	-	+		-	-	+	-	-	-	-	+	-	+	65-70	1:5,000	3-4

[a] + = infectious; - = not infectious.
[b] Figures in parentheses indicate citation to literature from which data was obtained.
[c] No symptoms produced but virus recovered.

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FIELD SPREAD OF RING SPOT VIRUS OF STONE FRUITS FROM SQUASH TO SQUASH[1]

J. A. Milbrath[2] and K. G. Swenson[3]

Summary

The vector responsible for the spread of ring spot virus of stone fruits[4] has not been determined, but these experiments show that the virus can be acquired from Buttercup squash and can be transmitted to squash plants by some vector or other agent under field conditions. These findings will greatly facilitate further greenhouse studies involving the search for the vector of this virus. Transmission tests were made with some of the insect species found in the squash plots. None of these insects transmitted the virus to squash under the conditions used in these studies. In the field plots natural infection decreased with increasing distances from the inoculated plants.

INTRODUCTION

A comprehensive search is being made in Oregon for the vector of the ring spot virus from stone fruits. Many vector tests have been made by feeding the test insect on infected stone fruit trees and then transferring them to virus free trees. This virus is often carried in these plants as a latent or symptomless virus and subsequent tests to such indicator plants as Shiro-fugen (Prunus serrulata var. Shiro-fugen Lindl.) are necessary to complete the experiment. This often involves 12 to 18 months to complete a single experiment. The search for the vector for this virus would be greatly facilitated if herbaceous hosts could be used in place of stone fruit trees. Ring spot virus from sour cherry trees showing sour cherry yellows symptoms will readily infect Buttercup squash, Cucurbita maxima Duchesne[5]. The virus can be maintained in this host and can be readily transferred by mechanical inoculations. Different strains of the ring spot virus will produce distinctive symptoms which can be easily recognized, a matter of considerable value in field studies involving natural spread. Attempts to use cucumber and squash for vector studies in the greenhouse failed to produce infected plants with the many vectors used. Experiments were conducted in the field during 1957 and 1958 to determine whether the virus would spread from either infected stone fruit trees or infected squash plants to healthy squash plants under field conditions.

1957 FIELD PLOTS

A site for the squash planting was selected where natural spread of the ring spot virus had occurred from infected Montmorency to virus-free trees; therefore, a vector was known to have been in this area in past years. Two rows of Buttercup squash were planted in hills 6 feet apart, the first row under the overhanging branches of Montmorency trees infected with ring spot virus and the second 4 feet farther away. When the cotyledons were fully expanded, alternate hills in each row were inoculated with a strain of ring spot (RS 31) not present in the nearby Montmorency trees. Thirty-nine of the 46 inoculated plants developed the bright golden leaf pattern distinctive for this strain of ring spot. One other plant in the inoculated row which did not show symptoms at first became infected late in the season and may have

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[1]Technical Paper No. 1214. Oregon Agricultural Experiment Station.

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[4]There is now such confusion in the literature as to the name of this virus that for the sake of clarity it will be referred to as the ring spot virus of stone fruits, rather than using peach ring spot, necrotic ring spot, cherry ring spot or various other names that have been used.

[5]Milbrath, J. A. 1956. Squash as a differential host for strains of stone fruit ring spot viruses. Phytopathology 46: 638-639.

been another case of natural spread.

On October 2, 1957 a final record was made on this plot for natural spread of the virus. Three hills each had one plant showing the bright golden chlorosis similar to the inoculated plants and another hill had two such plants. This made a total of five of the 46 plants in the hills not inoculated which had become infected with ring spot virus.

The strain of virus from these naturally infected plants was compared in the greenhouse with the strain used in the inoculations made in the field plants in the spring, and the virus that was transmitted was found to be the same strain as the original inoculum. This demonstrated that some unknown vector of the ring spot virus could acquire the virus from squash plants and could transmit it to other squash plants under field conditions.

1958 FIELD PLOTS

Two additional experiments on field spread of the ring spot virus were performed during the 1958 season. One site was selected which was several hundred feet from the nearest ring-spot-infected stone-fruit tree. It was adjacent to a grassy roadside and near a field of mixed legumes and grasses. Seventeen hills of Buttercup squash were planted 6 feet apart in a single row parallel to the road. When the cotyledons were fully expanded the plants in hills 3, 6, 9, 11 and 14 were inoculated with the same strain of ring spot virus used in the 1957 experiments. All plants in the inoculated hills showed at least one infected plant by June 30 and most of the plants showed symptoms by July 22 (Table 1). The first non-inoculated plant observed with virus symptoms was on August 4, but the symptoms were well developed on about 12 inches of growth which indicated infection had occurred at an earlier date. By August 11 many of the non-inoculated plants were showing symptoms and final records were made September 23 (Table 1).

Table 1. Natural spread of stone fruit ring spot virus from infected squash to non-inoculated squash. Plot I. 1958.

Hill number	Number plants inoculated	Number with symptoms: June 30	July 7	July 14	July 22	Number of plants infected September 23, 1958
1	0					1/5
2	0					1/5
3	5	0	2	2	3	5/5
4	0					1/5
5	0					1/5
6	5	1	2	4	4	5/5
7	0					1/5
8	0					4/6
9	5	1	2	5	5	5/5
10	0					5/5
11	5	1	3	4	5	6/6[a]
12	0					2/6
13	0					2/6
14	4	1	2	4	4	4/4
15	0					1/5
16	0					1/5
17	0					0/6

[a]Non-inoculated late emerging plants apparently became infected naturally.

Twenty of the 64 plants in non-inoculated hills became infected by natural spread of the virus. In addition, five plants in the inoculated hills which did not show symptoms July 22 had become infected by September 23, and probably represent spread by the vector, since symptoms from mechanical inoculations should have developed earlier.

Another experiment was designed to determine the distance that the virus might be carried from the site of inoculum. This plot was placed near an experimental orchard of originally virus-free Montmorency sour cherries where 16 of the 46 trees had developed necrotic ring

spot symptoms from natural spread. The plot was bordered on the north by the orchard, on the east by a road with borders of grass and other weeds, and on the south and west by cultivated sweet corn. Buttercup squash was planted in seven rows parallel to the road. There were 12 hills in each row with the hills 10 feet apart in each direction. On June 11 when the cotyledon leaves were fully expanded all plants in the row adjacent to the road were inoculated with the same strain of ring spot virus used in the other experiments. All cultivation was parallel to this row, and the vines were trained parallel to the row to prevent overlapping of the diseased and normal plants. Irrigation was by overhead sprinklers.

Seventeen of the 53 inoculated plants showed symptoms by June 30, 25 by July 7, 32 by July 14, 34 by July 22, and 35 by July 29. When the final record was made on September 23, fifty of the 58 plants on this row had developed the bright chlorotic patterns of the ring spot virus (Table 2). The first non-inoculated plants showed symptoms by August 11, so it was difficult to determine how many of the plants in the first row became infected by the mechanical inoculation and how many by natural spread. The 15 plants that developed symptoms after July 29 probably were infected by vectors.

Table 2 summarizes the total number of plants which became infected, either by mechanical inoculation or by natural spread.

Table 2. Natural spread of stone fruit ring spot virus from infected squash to non-inoculated squash. Plot II. 1958.

Hill number	Number plants inoculated	Number of plants infected September 23, 1958 — Row number 1	2	3	4	5	6	7
1	3	3/3	1/4	0/3	0/2	0/3	0/1	0/1
2	6	5/6	0/3	0/3	0/5	0/3	0/3	0/5
3	4	4/4	1/5	0/4	0/1	0/3	0/4	0/1
4	3	5/5[a]	2/5	0/3	0/5	0/2	0/3	0/5
5	3	4/4[a]	1/5	0/4	0/1	0/5	0/3	0/3
6	7	5/7	0/4	0/4	0/7	0/5	0/6	0/3
7	6	6/6	2/5	0/5	0/6	0/2	0/6	0/4
8	4	3/4	0/3	0/3	0/5	0/4	0/0	0/6
9	5	2/5	0/2	0/2	1/6	0/4	0/3	0/4
10	4	4/4	0/3	0/4	0/4	0/4	0/4	0/2
11	4	6/6[a]	0/3	0/3	0/6	0/4	0/3	0/0
12	4	3/4	3/5	0/2	0/7	0/3	0/2	0/1

[a]All plants in Row 1 that were of suitable size were inoculated, but some late emerging plants were not. Some of these became infected from natural spread.

Ten of the 47 non-inoculated plants in row two became infected with ring spot, apparently by vectors, and one of the 55 plants in row four became infected. None of the plants in rows three, five, six, or seven developed any symptoms indicative of virus infection.

INSECTS FOUND ASSOCIATED WITH SQUASH AND CERTAIN PRUNUS SPECIES WHICH MIGHT BE VECTORS OF THE VIRUS

The insect population associated with the 1958 squash plots was studied throughout the growing season. No aphids were colonizing on the squash plants during the part of the growing season when transmission occurred, but colonies of Aphis fabae Scop. were found late in the season. Several leafhopper species were found in the plots. Empoasca filamenta De L. was abundant and breeding on the plants. Draeculacephala crassicornis (Van D.), Macrosteles fascifrons (Stal.) and Euscelidius variegatus (Kbm.) were common in the plots, as was the spittlebug, Philaenus leucophthalmus (L.). Small numbers of Colladonus montanus (Van D.) and Endria inimica (Say) were found consistently in the plots, as was an unidentified species of Delphacidae.

Transmission tests were made with certain insect species which occur on squash or on Prunus species (Table 3). Beetles, leafhoppers and spittlebugs were placed on small squash plants which had been inoculated mechanically with the same strain of ring spot (RS 31) used

in the field plots. After 24 hours, they were transferred to healthy squash plants in the cotyledonary stage of growth. Aphid-infested leaves were placed on diseased squash plants and left overnight. By the following morning many of the aphids had crawled onto the leaves of the diseased plant from which they were transferred to healthy plants. Thrips were taken from diseased squash plants in the field and placed on small healthy plants. All insects tested were left on healthy squash plants for as long as they survived there. No transmission of ring spot virus was obtained in these tests.

Table 3. Insect species used in transmission tests with peach ring spot virus from Buttercup squash to Buttercup squash.

Species	Number individuals	Number plants[a]
Aphids:		
Anuraphis helichrysi (Kalt.)	2500	33
Aphis cardui L.	985	29
Aphis fabae Scop.	2000	40
Hyalopterus atriplicus (L.)	2000	40
Phorodon humuli (Schr.)	1375	55
Myzus cerasi (Fabr.)	1500	15
Myzus persicae (Sulz.)	500	38
Beetles:		
Diabrotica undecimpunctata Mann.	54	5
Leafhoppers:[b]		
Colladonus montanus (Van D.)	45	5
Deltocephalus grex Oman	105	12
Draeculacephala crassicornis (Van D.)	971	105
Empoasca filamenta De L.	242	19
Endria inimica (Say)	167	19
Euscelidius variegatus (Kbm.)	67	8
Macrosteles fascifrons (Stal.)	400	37
Spittlebugs:		
Philaenus leucophthalmus (L.)	137	27
Thrips:		
Several species	391	12

[a]In the case of leafhoppers and spittlebugs, this number includes only the first healthy test plant on which the insects fed. Many of these insects were subsequently transferred to series of several more plants.

[b]The writers are indebted to J. P. Kramer of the Insect Identification and Parasite Introduction Laboratories, Agricultural Research Service, for the identification of certain leafhopper species.

DISCUSSION

The discovery that an unknown vector has acquired the ring spot virus of stone fruits from mechanically inoculated squash plants, and has transmitted it to adjacent healthy squash plants, should greatly facilitate future vector studies with this virus. The rapidity of spread and the distinct symptom pattern makes squash a much more useful plant than the native hosts in the genus Prunus for study of field spread and behavior of this virus. Squash is also a better host for controlled greenhouse experiments.

These squash plantings have established small limited areas where the virus is known to spread, and this will restrict the number of insect species for future vector studies. Virus spread occurred in these plots during a 30 to 50 day period in July and August.

The pattern of virus spread in the squash was quite significant and certain speculations can be made from these results. In plot two of the 1958 plantings, ten squash plants became infected in row 2 which was 10 feet from the inoculated hills, and one plant in row 4 which was

30 feet from the inoculum. The inoculated plants were not allowed to come in contact with these plants, and the irrigation was by overhead sprinklers to avoid water or soil movement. The spread pattern and the short period of exposure before first transmissions in this plot would seem to eliminate the virus' moving through the soil or being carried by a soil inhabiting organism such as a nematode. A Prunus-specific eriophyid mite would not seem to be a probable vector since the virus was acquired from squash and transmitted to squash.

The high percentage of transmission in plot one, where the plants were 6 feet or less from the source of inoculum as compared with the relatively low transmission in plot two where the inoculum was 10 feet or more away, is difficult to interpret at this time, but does seem significant.

Virus transmission through pollen has not been eliminated by these studies and could be the method by which this spread has occurred. There were many honey bees and beetles (Diabrotica undecimpunctata Mann.) visiting or feeding on the squash blossoms throughout the blooming period.

OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS, OREGON, AND ENTOMOLOGY RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

POTATO VIRUS Y APPEARS IN ADDITIONAL AREAS OF PEPPER AND TOMATO PRODUCTION IN SOUTH FLORIDA[1]

John N. Simons

Abstract

During 1957 potato virus Y was found in both pepper and tomato plantings in areas of south Florida that previously had been free of the disease. The discovery of the disease in these areas followed, by 1 year, the introduction of commercial potato production. It seems likely the virus was introduced through infected seed potatoes.

During the period 1953-1956 it became apparent that the occurrence of potato virus Y in south Florida was restricted to certain areas[2]. Although the virus had been known in pepper and tomato from the Belle Glade, Homestead, and Bradenton areas for many years (Fig. 1A), it was unreported from either the East Coast or the Immokalee farming regions. The most obvious explanation for this situation was that potato virus Y occurred only where potatoes had been or were being grown on a commercial scale.

Since commercial potato production was initiated during 1956 in both the East Coast and Immokalee areas, it was predicted[2] that potato virus Y might very well appear in the pepper and tomato crops of these areas within a short time. During the Spring of 1957 this prediction was borne out with isolated cases of the virus being found in both areas. Last year, 1958, the virus occurred to such an extent in the East Coast section that considerable losses were reported (Fig. 1B). During the past growing season, 1959, the pepper crop along the East Coast has been afflicted with potato virus Y to a degree that has caused some growers to lose most of their crop. Estimates of an area wide yield reduction of over 50 percent have been made. Tomatoes, particularly the late (December-February) plantings, have suffered large losses also.

Interestingly, the tomato and pepper crops around Immokalee have not yet suffered greatly from potato virus Y. The explanation for this situation is in large part due to the fact that the farms in the Immokalee area are separated from each other by from 1 to 5 miles of virgin woods and swamps, while agriculture along the East Coast is much more intensive, with many farms adjacent to each other. Both areas are similar insofar as climate, presence of vectors and native host weeds, and general farming practices are concerned.

Undoubtedly part of the reason for the tremendous outbreak of potato virus Y along the East Coast this year was due to the early buildup of very large populations of the green peach aphid, Myzus persicae (Sulz.). Most farmers made no attempt to eliminate the principal weed host of the virus, nightshade, during the past summer. The result of this combination of factors was spectacular.

It seems evident from this 4-year history of the disease that the virus did arrive with the advent of potato production. The most tragic part of this story is that the value of the potato crop produced during 1956-1959 is at best a small fraction of the monetary loss caused by the virus in pepper and tomato plantings during either 1958 or 1959.

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[1] Florida Agricultural Experiment Station Journal Series, No. 900.

[2] Simons, J. N., R. A. Conover, and J. M. Walter. 1956. Correlation of occurrence of potato virus Y with areas of potato production in Florida. Plant Disease Reptr. 40: 531-533.

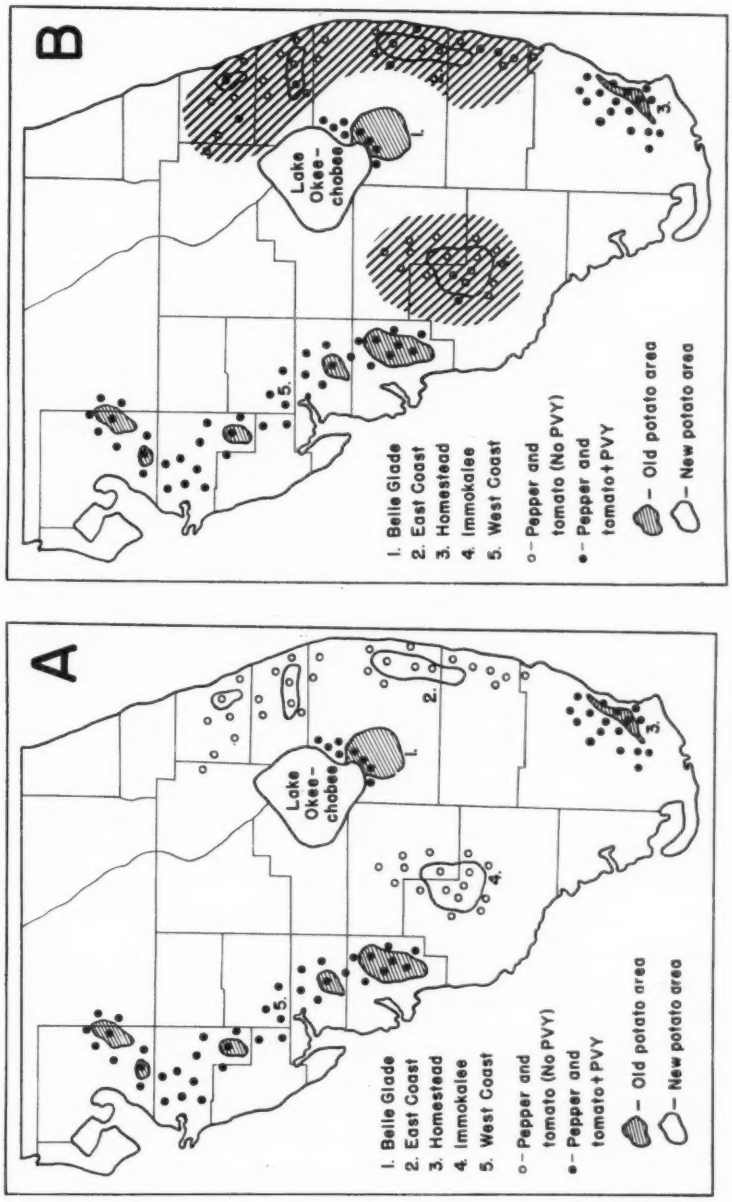

FIGURE 1. A -- Potato virus Y distribution in pepper and tomato prior to 1956. Note that the virus was found only where potatoes had been produced commercially. B -- Present distribution of the disease. Dashed lines indicate new areas of occurrence.

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A WHITEFLY, TRIALEURODES ABUTILONEA, AN INSECT VECTOR OF SWEETPOTATO FEATHERY MOTTLE IN MARYLAND

E. M. Hildebrand[1]

Abstract

A whitefly, Trialeurodes abutilonea[2], was found to be a natural vector of sweetpotato feathery mottle virus in Maryland. This insect's vector activity was conditioned by close proximity to the sweetpotato plant of the Indian Mallow weed, Abutilon theophrasti, on which the insect breeds copiously and readily abandons only as the weed goes to seed in late summer.

In 1954 a systematic study was started to test native insects as potential vectors of the sweetpotato viruses associated with internal cork and feathery mottle. By 1956 (5) two aphid species, Myzus persicae (Sulz.) and Macrosiphum solanifolii (Ashm.), had been shown to be efficient vectors of internal cork virosis, but incapable of transmitting feathery mottle.

All the probable vector types prevalent in one isolated sweetpotato planting were included in these studies. In tests with leafhoppers, 10 or more insects of each species were collected and placed on caged diseased plants for about 1 day. This step was followed by transfer to healthy plants in series for one or more days or for the duration of the experiment. Three collections were usually made each week during the growing season. During 4 years' work none of the leafhoppers tested was found to be a vector of internal cork or feathery mottle.

The search for an insect vector of feathery mottle was stimulated by the finding of a small amount of natural transmission in the field (2). The search went unrewarded until 1957 when a whitefly (7) was found breeding on Indian Mallow (Abutilon theophrasti Medic.) in the border of the planting. This tiny insect (presumably Trialeurodes abutilonea (Hald.)) was first found living on sweetpotato plants after the weed host began to go to seed and to drop its leaves. This insect was tested on a limited scale late that season, but with indifferent results. However, after the roots harvested from these tests were put through the "flush of growth" in the greenhouse (2) the sprouts from about 10 percent of them showed feathery mottle symptoms.

In October 1957, Sheffield (9) reported that virus B, which resembles feathery mottle, and is the more important of two sweetpotato viruses in East Africa, was generally transmitted by the whitefly Bemisia tabaci (Genn.), but that its performance as a vector was very inefficient.

In June 1958, Girardeau (1) announced that the sweetpotato whitefly (Bemisia inconspicua (Q.)) is the vector of sweetpotato mosaic in South Georgia, which virus is the same as feathery mottle (4). According to Russell (8), Bemisia inconspicua is a synonym of B. tabaci. As pointed out by Hildebrand (4), two syndromes are caused by sweetpotato viruses in the United States, Israel and East Africa. The aphid is a natural vector of the cork syndrome (6) and the whitefly of the feathery mottle ingested virus syndrome (9).

The 1957 season at Beltsville, Maryland was unusual. An August drought was followed by an abundance of rain and temperatures above normal in September. The appearance of feathery mottle symptoms on current-season sweetpotato growth that season was attributed to a climatic aberration (3). As it was suspected that a vector of feathery mottle might breed on wild plants, weeds were first allowed to grow undisturbed on the borders of the 1957 test planting. This brought the Indian Mallow weed, the host of the whitefly, in close proximity to the sweetpotato plant and was correlated with an increase in natural transmission, from 6 percent of the plants in the plot in 1956 to 28 percent in 1957.

The failure to get current-season feathery mottle symptoms more often than one year in three (1955-57) at Beltsville posed the need for detailed knowledge on the biology of the whitefly, to establish its possible vector pattern. Such investigations were begun in 1958 in the field and

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[2] The 1958 insect vector was identified by Louise M. Russell, Entomologist, Insect Identification and Parasite Introduction Laboratories, Entomology Research Division, Agricultural Research Service, United States Department of Agriculture, Washington, D. C. The whiteflies observed in 1957 presumably were T. abutilonea, but none were submitted for determination.

under laboratory and greenhouse conditions.

In 1958 whiteflies were first noted in the field in early July, at about the 4-leaf stage of the mallow weed. Cool rainy weather generally retarded plant growth into July. Because of the favorable weather that followed, good vegetative growth continued almost to the end of the growing season. Transplanting the mallow from the border of the field into the sweetpotato planting in midsummer failed to induce the whiteflies to leave the weed in significant numbers. Late in the season when the weed became defoliated and went to seed, the whiteflies moved off the mallow. As in 1955 and 1956, no current-season feathery mottle symptoms developed in 1958.

The life habits of the whitefly on the mallow host were studied simultaneously in the field and under controlled conditions on mallow, sweetpotato, and morning glory in the laboratory and greenhouse. This insect much preferred the weed as a host but bred well on two morning glories (Ipomoea alba and Quamoclit angulata). It lived relatively poorly on sweetpotatoes; however, it reproduced well enough on the clones employed in these studies to permit the conduct of over 100 transmission experiments.

The life cycle, or one generation, consisted of the egg, three larval stages, the pupal stage, and the adult. Only the first larval "crawler" stage was mobile and then but for less than 24 hours, after which its stylet, arising in the rostrum, penetrated the leaf into feeding position where it remained permanently. The larval stages and emerging adult leave this original stylet in situ and the adult develops a proboscis with a new stylet. After coming to rest the "crawler" larva lost its mobility. Subsequently it was impossible to transfer this, or the other immature stages successfully, but it was possible to transfer the adult. Under the several conditions encountered in the laboratory, greenhouse and field, each generation of this insect ranged between 18 and 24 days during midsummer, and each of the six stages lasted for 3 to 4 days.

Numerous experiments, involving the use of six large cages, demonstrated that adults of the whitefly (T. abutilonea) transmitted feathery mottle virus in the greenhouse. This vector readily transmitted the virus from diseased sweetpotato and morning glory to healthy sweetpotato and morning glory. However, the inoculations frequently failed to induce current-season symptoms unless the host was in the proper growth status, which was true in only about 20 percent of the trials.

The best transmission procedure was to have a whitefly colony established on diseased sweetpotatoes in a cage large enough to accommodate several healthy plants. Healthy plants were then moved into the cage for intervals varying from a day to a week. The adults emerging from pupae on diseased leaves were viruliferous and almost immediately started to feed on healthy plants and transmit the virus that had been ingested during immature stages of the insect.

To demonstrate rapid transmission, three factors must be synchronized: 1) emergence of viruliferous adults from pupae on diseased foliage; 2) the proper stage in development of the indicator plant; and 3) a growth-status of the indicator plant characterized as a "spurt of growth" as inoculation takes place.

Several rapid transmission experiments, in which the essential factors were synchronized, demonstrated that after an incubation period of 11 to 15 days the whitefly is an effective vector of feathery mottle virus on indicator plants. This period corresponds closely to the shortest incubation period (10 to 14 days) noted for graft transmission of the virus on sweetpotato indicator plants.

The variability in transmission efficiency (from 0 to 100 percent) of the feathery mottle virus by the whitefly can possibly be attributed to the intricacy of the insect's life cycle, plus the variable character of the plant suscept response to inoculation with the virus. The results of the work clearly show that the whitefly, which occurs in all States where the sweetpotato is grown, is an effective vector of feathery mottle virus when the factors essential for transmission are synchronized.

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IMPORTANCE OF MICROSCOPIC OPENINGS IN VECTOR TRANSMISSION OF PLANT VIRUSES AND BACTERIA

E. M. Hildebrand[1]

Abstract

Recent studies of numerous inoculation mechanisms support the hypothesis that for virus infection of epidermal cells of plants there is, in most instances, a simple elementary mechanism involving: 1) production of minute microscopic wounds (< 6μ in diameter), 2) the simultaneous pulsing action of protoplasm through the wound opening for virus entry (6/10 second), and 3) prompt healing to insure survival of host cell and virus. The same criterion for wound size also seems to apply when ingested viruses are introduced into the phloem cells by deep-feeding arthropods.

As a result of micrurgical studies initiated in 1937 and still in progress, it has been experimentally established that precise microscopic openings are the common mode for entry of obligate parasitic entities in vector transmission of plant viruses. It is evident that the production of microscopic wounds is the first essential for vector action in certain modes of plant virus inoculation (12, p. 81). The term "vector" herein employed conforms to the general pathology definition of Reyniers (19), "The routes of transfer are called vector lines, and the objects upon which these organisms are transferred are the vectors....All vector lines may be classified as contact or air-borne."

The details of the preliminary micrurgical studies on bacterial and virus infection were not published. The size of wound safe for host cell survival was first established in microinjection studies with two bacterial species, the fire blight (Erwinia amylovora) and the crown gall (Agrobacterium tumefaciens) organisms. This was done before initiating work on viruses in 1938.

Ordinarily bacterial pathogens are located intercellularly at the site of inoculation where, under favorable conditions, they multiply rapidly. When in the apple flower nectary, fire blight bacteria may increase from one to a million cells within 24 hours, preliminary to establishing infection (10). It was found that any natural opening (such as a nectar-secreting stomate and the open intercellular spaces on the stigma of flowers) or artificial opening (such as a mechanical or insect puncture) into the intercellular spaces functioned as an infection court for the fire blight organism. Single crown gall bacteria readily produced infection intercellularly in needle-puncture wounds, but when this wound parasite was inoculated into living cells of the tomato (Fig. 1, A), the bacteria failed to survive in the intracellular environment (12, pp. 84-88). This is in direct contrast to the viruses that infect only intracellularly.

The work with viruses was on the development of an inoculation technique that would ensure host-cell survival and at first was limited to studies with yellow aucuba mosaic virus through wounding of the trichomes on the intact stems of tomato and tobacco plants. By 1939, after thousands of wounds were tested, it was established that for host-cell survival, the wounds must be smaller than 6μ in diameter. When the tip portion of the micropipette was less than 3μ in diameter, over 75 percent of the wounded cells recovered compared with less than 40 percent survival when the tips were about 5μ to 6μ in diameter.

The mechanism of micropipette-wound transmission of tobacco aucuba mosaic virus, as originally observed in trichomes (12, p. 81), was as follows: "When the micropipette perforates the trichome wall, protoplasm gushes out to form a hemispherical droplet which bathes the pipette tip momentarily and then returns into the cell." The speed of this pulsing mechanism was reported to be consistently about 6/10 second (15). Based on subsequent studies of several modes of virus transmission it appears that this mechanism probably applies to all vector lines: contact (arthropod feeding and other wounding) and air-borne (such as pollen transmission with entry through natural openings). In recent studies on the mechanism of

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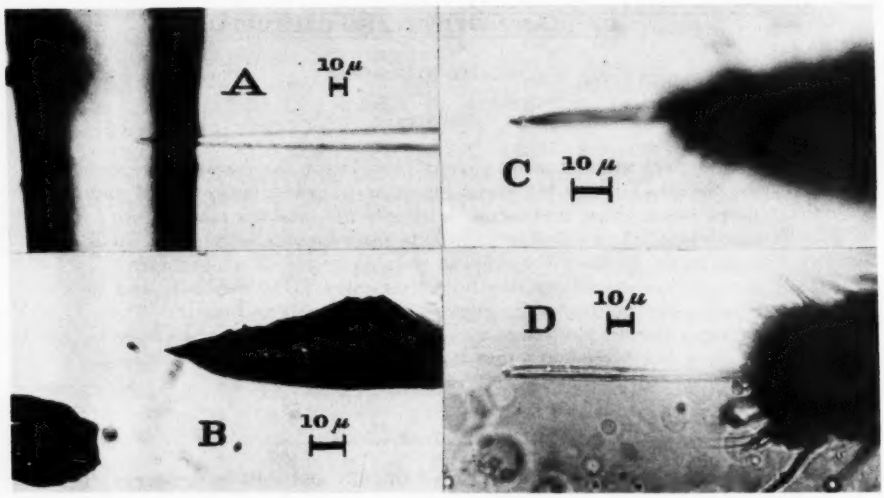

FIGURE 1. Some modes of host cell perforation in the transmission of plant viruses. A -- a 3μ micropipette immediately after inoculation of trichome on tomato stem with yellow aucuba mosaic virus. B -- a sharp 320-mesh carborundum particle with the 10μ of the tip portion <5μ in diameter. C -- stylet of the aphid Myzus persicae, vector of sweetpotato cork virus, with the first 15μ of the tip portion about 2.6μ in diameter. D -- stylet of the leafhopper Scaphytopius acutus, vector of peach yellow-red virosis, with the tip portion about 3μ in diameter.

plant virus inoculation the unstable, labile sweetpotato cork virus was employed instead of the stable aucuba mosaic virus used in the earlier work.

In general, vector transmission of labile plant viruses, which ordinarily are inactivated in vitro, apparently entails an extremely delicate response to wounding, by whatever means. It probably involves the swift entry of virus in contact with and as a film on the momentarily extruded protoplasm of the inoculated cell. Inoculation with stable viruses, which survive for a time in vitro, or outside the living cell, may be effected either through slight non-fatal wounds in the cell including those by sucking insects or less commonly through severe mechanical or chewing-insect wounds that are fatal to the host cells. Entry here into the adjoining living cells is possible because there is an interval of exposure of several minutes after such wound inoculations during which the virus probably moves from its location in the protoplasm of the dying cell, via the plasmodesmata, into adjoining living cells before the opening or pores in the latter cells are plugged by coagulated protoplasm.

Mechanical transmission is often apparently synonymous with micro-wounding as exemplified in micropipette inoculation, already discussed. The most important character of the new "squeeze-rub" technique of mechanical inoculation used for rapid transmission of the sweetpotato cork virus (14) is that wounding (rubbing with carborundum or not) and virus entry are coincidental, something which is essential for cork virus survival. This technique apparently introduces the virus into the host cells in a manner that obviates contact with air, a thing that the aphid vector of the cork virus appears to do naturally. Apparently the interior of the aphid proboscis temporarily protects its retracted stylet and any virus thereon from exposure to air after each probing puncture. Besides, the aphid proboscis excludes air when it is appressed on the feeding surface before exserting its stylet again. Once inside the epidermal cell the virus multiplies rapidly and moves slowly through several parenchyma cells (probably via plasmodesmata) into the phloem where it is rapidly translocated in the food channel and thence into young developing leaves for symptom expression.

Carborundum and other abrasives are often used in the rubbing technique by man (the vector) to increase or expedite mechanical inoculation with viruses. However, only a very small percentage of the particles in carborundum abrasives of 320 and 600 mesh are adapted

for safe perforation wounding (<5μ in diameter) of epidermal cells to effect inoculation with stable viruses like aucuba-mosaic or labile viruses like sweetpotato cork. Figure 1, B illustrates a carborundum particle of proper dimensions for host-cell survival. Rubbing distributes virus particles all over the inoculated surface. The resultant injuries may include virus entry into larger wounds that kill the host cells. Thus within a limited time it is possible for a stable virus to move from the dying cell into adjoining living cells through plasmodesmata before they are plugged.

The stylet of the green peach aphid, Myzus persicae (Sulz.), a vector of the bacteria of the fire blight organism (11) and also of numerous viruses, was found to approximate 3μ in diameter when first measured in New York in 1939. More recently measurements were made of adults and nymphs of different instars of M. persicae and all were found to approximate 2.6μ from the front view and 2.1μ from side view (Fig. 1, C). That the aphid functioned as a virus vector was first indicated by Doolittle and Walker (9). According to Snodgrass (20, p. 341) the aphid, per se, has three feeding postures or stances which can be interpreted as probing, shallow-feeding, and deep-feeding. The shallow-feeding posture was illustrated on pomaceous fruits (11), when the aphid was functioning as a vector of fire blight bacteria. Aphids were first reported to be natural vectors of sweetpotato root cork virus in 1956 (16) as a result of studies begun in 1954 and published in detail in 1958 (17). The studies showed that two species, (M. persicae and Macrosiphum solanifolii), after being starved for 2 hours picked up and readily transmitted the sweetpotato cork virus from diseased leaves of several infected hosts to susceptible hosts.

The position of potato virus Y on the stylet tips of aphids was well established by Bradley and co-workers in Canada (1-6). Similar results were reported by Day and Irzykiewicz (7-8) in Australia on the mechanism by which so-called nonpersistent phytopathogenic viruses (a group which includes the sweetpotato cork virus) are transmitted by aphids. According to their working hypothesis, "The stylets of the vector become contaminated with virus during probing in infected tissues. Only this virus is of significance in virus transmission and ingested virus plays no part in the process." The work herein reported on the position of the sweetpotato cork virus on the stylet tips of aphids conformed with the above findings in Canada and Australia.

The characteristic behavior of M. persicae on plants susceptible to cork virus was demonstrated to range between the probing and shallow-feeding stances with stylet penetration varying from 8μ to 35μ. Immediately upon arrival upon a site the aphid started to probe, both when picking up the virus from diseased leaves and when inoculating virus into healthy leaves. Between 10 and 20 percent of the individual aphids (nymphs or adults) probed to the right depth or only into the epidermal layer to effect inoculation. The interval both for picking up and for depositing this virus was short and ranged from about 10 seconds to 10 minutes. The depth of the probing and shallow feeding which prevailed at a given moment was determined by chloroform asphyxiation of the insects in situ. This treatment prevented them from retracting their stylets and thereby made it possible for examination to reveal the actual depth of feeding. On the basis of numerous tests where infection resulted, the average penetration of the stylet tips in early probing appeared to be about 15μ to 20μ. When inoculated by aphids or mechanically and once within the epidermal cells of Scarlett O'Hara morning glory, the cork virus multiplied rapidly, moved slowly through several parenchyma cells (probably via plasmodesmata) into the phloem and from there was rapidly translocated about 7 inches into parenchyma cells of young developing leaves, resulting in symptom expression within 5 to 7 days.

There is a dearth of information on the manner in which deep-feeding arthropods function in virus transmission, but one thing is clear -- only ingested virus can be transmitted in this way. Ingested viruses largely reside in the translocation stream in the phloem tissue. The deep-feeding vectors must pierce phloem cells to tap the translocation stream for their food supply, and it is in this way that they become viruliferous. The vector of the peach X-disease virus was discovered in 1942 (13). The stylet tip of the insect vector (Scaphytopius acutus Say.) was found to measure about 3μ in diameter (Fig. 1, D), a size definitely safe for wounding phloem cells. This size is not distinctive from other non-vector types but is smaller than for the less efficient vector (Colladonus clitellarius Say.) of the same disease, which leafhopper had a stylet tip diameter ranging between 5μ and 8μ. Recently Pollard (18) reported on a histological study of the whitefly vector (Bemisia tabaci Genn.) of the cotton leaf curl virus. He illustrated with a camera lucida drawing the feeding position of the insect stylet in a phloem cell. When the literature was found devoid of information on the precise size of stylets of the arthropod vector types, numerous specimens were studied micrurgically.

Aphids, leafhoppers, whiteflies, mites, and nematodes were tested on a limited scale and found to possess stylets with tip diameters conforming to the size criterion for transmission. Even though some had stylets of a size to effect non-fatal penetration of cells, none of the leafhoppers tested at Beltsville between 1955 and 1958 were vectors of either of the sweet-potato virus syndromes (internal cork or feathery mottle). In other words, factors other than size of stylet determine the vector efficiency of a given insect. Studies on this subject are continuing.

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TOMATO DISEASE CONTROL IN RELATION TO GALLONAGE AND FUNGICIDE APPLICATORS[1]

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Abstract

Field experiments over a period of 5 years and under disease conditions ranging from mild to severe indicated that comparable control of early blight and anthracnose of tomato was obtained with a hydraulic applicator at rates of 50, 100 and 200 gallons per acre; a commercial air-blast applicator at 50 and 100 gallons per acre; and an experimental concentrate type applicator, the Cornell Row Mister III, at 20 gallons per acre, provided the fungicide dosage per acre application was constant. The deposition and distribution of the fungicides by the applicators, determined by leaf print methods, supported the disease control data.

INTRODUCTION

The acceptance of a tomato spray program by growers and processors in New York State following the disastrous late blight epiphytotics in 1946 and 1947 soon led to numerous inquiries about methods of application. Of particular interest were questions relative to fungicide concentration, gallonage, and the suitability of modified orchard air-blast equipment for tomatoes. The most serious objection to the use of the hydraulic row crop applicators was the large volume of water recommended per acre application--namely, 200 gallons. That factor entailed considerable expenditure of time and money in hauling water and the inability to cover many acres per hour at the required slower speeds. In addition, the tendency of growers to apply lower than recommended gallonages in the early sprays when plants were small and higher gallonages when the plants became large disrupted the fungicide dosage schedule, which was based on a given amount of material per 100 gallons at a 200 gallon per acre application rate.

In order to clarify some of these problems, experiments were conducted in 1951 through 1955 to compare different gallonages with hydraulic equipment at a constant fungicide dosage per acre application and to evaluate air-blast applicators at reduced gallonages but with constant fungicide dosages. Disease control and, in some instances, fungicide deposition and distribution as determined by leaf prints were used as criteria. The data are presented by years because treatments and equipment varied from year to year according to progress and availability of equipment.

MATERIALS AND METHODS

Tomato varieties, fungicide schedules, applicators, gallonages and experimental plot designs varied somewhat with the years and are therefore described briefly under the results for each season. Each applicator was calibrated to deliver the desired gallonage. The standard of comparison was a hydraulic machine that applied 200 gallons of spray mixture per acre at 600 p. s. i. pressure through six nozzles per 6-foot row width, each nozzle containing a number 3 disc and located 1 foot apart on the boom. Drop pipes were used in the first three applications. Succeeding applications were made through a brush-type boom.

In 1951 and 1952 experiments were designed primarily to determine whether a given amount of fungicide per acre application could be applied just as effectively in 100 gallons or less, as in 200 gallons per acre. In 1953 the object was to compare the hydraulic machine with an experimental machine, the Cornell Row Mister III, which delivered the same amount of fungicide in only 20 gallons per acre. The Cornell Row Mister III is a concentrate type sprayer equipped with a unique rotating-turret-head distributing system consisting of four round outlets rotating in a horizontal plane. High velocity air is discharged through these outlets during the forward 180 degrees of rotation. Low pressure liquid is injected into the air stream during the forward 120 degrees of rotation. The resulting swath pattern is laid down in a series of overlapping cycloidal curves as the sprayer moves forward.

[1] Approved by the Director of the New York State Agricultural Experiment Station as Journal Paper No. 1163.

In 1954 the standard hydraulic machine was again compared with the Cornell Row Mister III and, in addition, with a commercial air-blast machine, the Bean Speed-Aire. The latter machine was calibrated to deliver the fixed fungicide dosage in 100 and in 50 gallons per acre. The Bean Speed-Aire is a dilute air-blast sprayer with a two-sided delivery. Medium velocity air is directed at right angles to the fan through a rectangular slot-type opening on each side of the sprayer. High pressure liquid is injected into the air stream immediately adjacent to the air outlet and the resulting spray laden air is carried out over the treated surface perpendicular to the direction of travel.

In 1955, a larger model of the Cornell Row Mister III, namely, Row Mister IV, was compared with the standard hydraulic machine in non-replicated strips in several commercial fields. Applications by the various machines were made on schedule, even when the wind velocity was as high as 12 miles per hour.

Leaf Print Methods

The deposition and distribution of the copper and maneb fungicides on the plants were determined by leaf prints. All leaf print procedures have three essential parts. First, the substances sought must be dissolved and absorbed on a white surface. By pressing each sampled leaf between a folded filter paper moistened with a suitable solvent, a minutely exact pattern of the spray deposit on both upper and lower leaf surfaces is transferred to the paper. Next, a highly colored compound must be developed from the adsorbed copper or carbamate ions. Thirdly, the colored print must be fixed or stabilized to form a permanent record. Deposit ratings on an arbitrary scale ranging from 0 to 10 were scored by at least three individuals working independently. For the present purpose, this distributional type of analysis was more meaningful than the single average deposit per unit area obtained by ordinary chemical methods. Individual deposit ratings were subjected to statistical analysis and used as criteria of the uniformity of deposition and overall performance of the applicator.

Ten leaves were selected at random from each of three positions on the plant; namely, top, inner middle, and inner bottom. Within a given experiment leaf samples were collected as soon as the spray had dried, and again after a weathering period. A letter press was used to transfer the deposit to the paper. A good grade of hardened filter paper (Schleicher and Schuell #575 or equivalent) was used. Each paper was identified with a code number in India ink before use. The procedures for the two fungicides are as follows:

Copper Prints: Stable reagent paper is prepared in advance by immersing in a 0.1 percent solution of dithiooxamide in 95 percent ethanol and drying. The solvent solution contains 50 gm ammonium nitrate, 150 ml concentrated ammonium hydroxide, and distilled water to make 1 liter. Press the leaves for 30 seconds, rinse in running water 30 minutes and dry.

Maneb Prints: Press leaves for 1 minute between papers moistened with a solvent solution containing NaOH 50 gm, 95 percent ethanol 100 ml and distilled water to make 1 liter. The developing solution contains $CuSO_4 . 5H_2O$ 10 gm, 100 ml of glacial acetic acid and distilled water to make 1 liter. Develop prints in this solution no longer than 10 seconds to avoid fading. Dip immediately for 10 seconds into a fixing solution of 5 percent aqueous sodium bicarbonate. Maintain the fixing solution at pH 7 to 8 by adding more bicarbonate as needed. Rinse in running water 30 minutes, and dry.

RESULTS

1951 Season

Fungicides for the three schedules, alternating ziram-bordeaux (Z-Z-B-Z-B), maneb, and captan were applied with a hydraulic machine at dosages of 4-16/8 (Z-B), 4, and 6 pounds per acre application, respectively, in 100 and 200 gallons. Plots of the above treatments consisted of 10-plant single rows bordered on either side by non-sprayed buffer plots, and together with a non-sprayed check plot were arranged in a 7 by 7 Latin Square design.

Statistical analyses of the data on early blight defoliation, anthracnose, and yield indicated no significant differences except those between the check plots and the fungicides at either gallonage. By excluding the check plots the experiment was analyzed as a factorial involving fungicides and gallonages, but no significant differences were obtained with any of the data except for one interaction between fungicides and gallonage that was due to a slight reversal in order of anthracnose development in one fungicide schedule from that of the other two (Table 1).

Table 1. Summary of tomato disease control and yield obtained with three different fungicide schedules applied at constant dosages per acre in different gallonages, 1951 and 1952, Geneva, New York[a].

Gallons Per Acre	Pressure p.s.i.	Early Blight Defoliation Per cent 1951	1952	Anthracnose Per cent 1951	1952	Yield of #1 Equivalents Tons/Acre 1951	1952
200	600	2	8	5.5	3.5	32.0	17.9
100	600	2	7	5.1	3.1	32.7	18.0
50	400	-	9	-	3.5	-	17.8
50	300	-	9	-	3.1	-	17.9
Level of Significance		NS	NS	NS	NS	NS	NS
Check	-	85	77	21.3	6.7	25.4	15.3

[a] No significant interactions occurred between fungicide and gallonages, or any that would affect the influence of gallonage, nor was there any influential difference between fungicides.

1952 Season

The same treatments that were used in 1951 were included in 1952 but, in addition, the fungicides for the three schedules were also applied in 50 gallons per acre, in one treatment at 400 p. s. i. pressure and in another at 300 p. s. i. Application of 50 gallons per acre was achieved through the use of No. 8001 SS TeeJet nozzles. Plots were of the same size as those in 1951, but they were arranged in random order in each of six blocks.

Leaf samples were taken for leaf prints from the ziram-bordeaux plots approximately 4 hours after the third application, which was bordeaux, to compare the deposition and distribution of the fungicide at the different gallonages. Ten days later, during which time precipitation ranged from 0.07 to 0.19 inches on four different days, another set of leaf samples was printed to compare the effects of weathering. Statistically, the deposit ratings on the upper surfaces of the non-weathered leaves were essentially the same for all gallonages, regardless of the plant position. The 50 gallons per acre treatments, however, deposited a smaller amount of fungicide on the under surfaces of the leaves, especially on those samples selected from the inner middle and inner bottom position of the plant. Data from the weathered samples were of the same order, but the ratings were lower. That was due in part to wash-off, to fungicide redistribution and to sampling error resulting from a change in stature of the plant.

Disease development was rather mild in 1952, but highly significant differences were obtained between the check plots and those that received the fungicide applications. No significant differences were obtained in yield. Analysis of the factorial part of the experiment that involved gallonages and fungicide schedules showed that equally good control of early blight defoliation and anthracnose were obtained with the fungicides, regardless of whether they were applied in 50, 100, or 200 gallons per acre (Table 1). A slight but significantly better control of anthracnose was obtained with the captan schedule than with the ziram-bordeaux.

1953 Season

The Cornell Row Mister III was compared with the standard hydraulic machine, using two fungicide schedules at each of two locations, Seneca Castle and Sodus, New York. The design of the experiment was a half-plaid Latin Square. The two fungicide schedules (and a check plot) were used as split blocks. The applicators (and a check plot) were arranged in a 3 x 3 Latin Square for each schedule. Each plot consisted of 9 rows, 75 feet long. The center row was planted to the Gem variety and the four rows on either side were planted with Red Jacket,

Data were obtained separately from the center row of Gem and the combined fourth and sixth rows of Red Jacket. Six applications were made at each location. Maneb at 4 pounds per acre application constituted one schedule, and ziram at 4 pounds and a fixed copper at 8 pounds per acre application in the order, Z-Z-C-Z-C-Z, the other. The materials were applied with the Row Mister III at 20 gallons per acre and with the hydraulic machine at 200 gallons.

Leaf prints were made from samples collected at the end of the third application in the copper-sprayed plots. Ten days later, during which time 1.5 inches of precipitation occurred over a 4-day period, another set of samples was selected from the same plants to determine the effect of weathering. No significant interaction between location and applicators existed in deposit ratings for either sampling date. Initial deposits of copper on the upper and lower leaf surfaces at the top position of the plants sprayed with the Row Mister III were significantly greater than those from plants sprayed with the hydraulic machine (Table 2). No significant

Table 2. Copper deposit ratings on leaves selected from three positions on plants from applicator plots treated with ziram-copper schedule, Sodus and Seneca Caste, New York, 1954[a].

	Leaf Position on Plant[b]					
	Top		Middle		Bottom	
Applicator	U	L	U	L	U	L
Row Mister III	7.5**	4.2*	4.6	1.2	1.1	0.3
Hydraulic	4.8	2.7	3.8	1.3	1.6	0.3

* Significant at odds of 19:1
** Significant at odds of 99:1
[a] Deposit ratings range from 0 (no deposit)to 10 (complete).
[b] U - Upper leaf surface.
L - Lower or underside of leaf.

differences between applicators were obtained at the other two plant positions. Copper residue analyses, based on micrograms of copper per 100 leaf punches, of similarly selected leaf samples confirmed the data from the leaf prints. Deposit ratings from the weathered samples were considerably lower than the initial deposits, especially at the top plant position, but they showed the same relative differences between applicators.

Diseases, in general, were more severe at Seneca Castle. The maneb schedule gave significantly better disease control at both locations than the ziram-copper schedule. No significant interaction, however, existed between schedules and locations, nor between location and applicators with respect to early blight defoliation, Alternaria fruit rot or anthracnose. Development of the above diseases was significantly less in the plots treated according to the two fungicide schedules, irrespective of applicators, than in the check plots. No significant differences in disease development occurred between the Row Mister III and the hydraulic applicator (Table 3).

No significant yield differences could be attributed to applicators at either location. In fact, none existed between the check plots and the treated plots, but that was due largely to the fact that another picking should have been made but circumstances other than weather prevented it. Had this been done, the yields would have been substantially greater in the treated plots, for the check plots were completely destroyed by disease; whereas, the treated plots were loaded with harvestable fruit.

1954 Season

The Row Mister III and the hydraulic applicator, as used in the 1953 season, were compared with the Bean Speed-Aire at 50 and 100 gallons per acre application, using only one fungicide schedule, namely, maneb at 4 pounds per acre application at one location, Seneca Castle. The four treatments together with the check plots were arranged in a 5 x 5 Latin Square. Plot size, variety arrangement, and the number of applications made were the same as in 1953.

Table 3. Summary of tomato disease control in varieties Gem and Red Jacket obtained with a constant fungicide dosage per acre applied with two machines at vastly different gallonages in two locations, 1954[a].

Applicator	Early Blight Defoliation (Per cent)				Anthracnose (Per cent)			
	Gem		Red Jacket		Gem		Red Jacket	
	S	SC	S	SC	S	SC	S	SC
RM III (20 Gals/A.)	16	34	12	18	2.4	14.6	2.4	4.4
Hydraulic (200 Gals/A.)	13	25	11	16	3.1	11.1	2.0	2.9
Level of Significance	NS	NS	NS	NS	NS	NS	NS	NS
Check	90	90	84	84	6.7	33.2	3.5	7.6

[a] Data represent average of two different fungicide schedules.
Locations: S -- Sodus, SC -- Seneca Castle, New York

Table 4. Disease control and yield of two varieties of tomato under constant fungicide dosage schedule applied in different gallonages with different applicators, Seneca Castle, New York, 1954.

Applicator	Gallons Per Acre	Early Blight Defoliation (Per cent)		Anthracnose (Per cent)		Tons/Acre #1 Equiv.	
		Gem	RJ	Gem	RJ	Gem	RJ
None	-	82	80	22.5	17.9	14.5	15.5
Hydraulic	200	6	6	3.8	3.7	18.1	19.5
Speed-Aire	100	7	7	4.7	5.0	19.2	21.6
Speed-Aire	50	7	7	5.9	3.3	16.4	17.2
RM III	20	8	6	8.5	3.7	17.3	18.4
LSD (19:1)		13	9	3.3	1.8	NS	NS
(99:1)		18	12	4.6	2.6	NS	NS

Leaf prints were made from samples collected at the end of the fourth application of maneb. Six days later another set of samples was collected for data on weathering. Those made immediately after the fungicide application showed no significant differences among applicators on the upper and lower surfaces of leaves from the top position of the plant. In the middle of the plant, the deposits were slightly less on the plants treated with the Row Mister III, which is in direct contrast to the copper deposits obtained in the 1953 experiments in which the Row Mister III was superior to the hydraulic machine. The slight discrepancy in 1954 could have been due in part to the fact that the nozzles on the Row Mister III became partially clogged during the fourth application and it was not noticed until the last plot was treated. Deposit ratings on the leaves from the bottom of the plants were about the same for all applicators. The weathered samples showed the same relative differences as the non-weathered except that the ratings were lower.

Early blight defoliation was moderate and, under these conditions, no significant differences attributable to the applicators were found (Table 4). Anthracnose was quite severe in the variety Gem, and the Row Mister III did a significantly poorer job in controlling the disease in this variety than the other two machines, although no differences in control were found among the applicators for the variety Red Jacket. The Bean Speed-Aire appeared to control the diseases as well when the material was applied in 50 gallons as in 100 gallons per acre. Disease control in all applicator plots was significantly better than in the check plots.

Although the yield differences among the three machines and the check plots were not significant for either variety, they did range from 1.7 to 6.1 tons more of number 1 equivalent fruit per acre than the checks.

1955 Season

The primary purpose of the experiments in this year was to demonstrate the comparative performances of the hydraulic machine, the Bean Speed-Aire, and the Row Mister IV, which is a larger model of the RM III and designed to cover a wider swath. Single replicate strips in each of two fields were treated with each of the three machines. The effective swaths and the gallonages of the hydraulic, Speed-Aire and RM IV were 18, 50 and 36 feet, and 200, 50 and 20 gallons, respectively. Data were obtained from marked-off sections in each plot. All three applicators performed equally well against early blight defoliation at both locations, there being from 7 to 12 percent defoliation among the treated plots, compared with 94 percent in the nontreated check strips. Anthracnose control was the same for all applicators at one location but at the other there was some indication that control was poorer in the strip in which the RM IV was used. The discrepancy is not entirely explainable, but it could have been caused by the location of the strip within the field and the lack of sufficient replication to measure such effect. No yield data were taken.

DISCUSSION AND CONCLUSIONS

Disease control data, obtained under conditions ranging from mild to severe, and fungicide deposition and distribution data, as determined by leaf prints, indicated that gallonage is not necessarily the main determinant of a good tomato fungicide schedule, provided the fungicide dosage per acre-application is constant. These results should not be interpreted as a blanket endorsement of any or all types of spray concentrate machinery for tomatoes, but they do indicate that the principle of concentrate application of tomato fungicides is sound. The greatest deterrent to their successful use would be the tendency of operators to over extend the limits of coverage with a particular applicator. The use of leaf prints is a useful method to determine the limits of a particular applicator under the range of conditions, especially wind velocity, normally encountered for a given locale.

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AN EVALUATION OF SPRAY MATERIALS FOR CONTROL OF BACTERIAL SPOT ON FIELD SEEDED TOMATOES

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Summary

New and old materials applied as sprays were tested for control of bacterial spot on field seeded tomato plants in three experiments during the fall of 1958. Copper materials and streptomycin provided good control under relatively mild conditions. Copper plus streptomycin tended to give the best control.

INTRODUCTION

Bacterial spot (Xanthomonas vesicatoria (Doidge) Dows.) has caused more damage to tomato plants in south Florida in recent years than any other disease. This disease is most severe during the rainy season, August through October. However, bacterial spot can develop after heavy rains during any month of the year.

One of the reasons for widespread damage by bacterial spot is that present methods do not give adequate control. Certain chemical seed treatments and fermentation extraction of seed have not provided adequate control of the disease. Rotations probably would not be beneficial, since field seeded crops on virgin soil usually become as severely diseased as those on continuously farmed land. Spraying on a rigid schedule with copper and streptomycin has provided the best control of bacterial spot (3, 4). However, such a schedule cannot be practically followed by most farmers throughout a crop.

Continued experimentation with spray materials seems to be the most rewarding method of improving control of bacterial spot. New and old materials were tested in three experiments during the fall of 1958.

MATERIALS AND METHODS

A randomized complete block design was used in all spray trials. Standard cultural methods used in the Indian River area were followed. Insecticides were applied separately as needed. The Indian River variety of tomato was planted in all tests. This variety is resistant to gray leaf spot which can be confused with bacterial spot because of the very similar symptoms.

Materials were applied with the aid of a trailer-mounted power sprayer, which developed 350 pounds pressure per square inch at the pump. Three to seven nozzles, depending on plant size, equipped with No. 3 discs were used to cover each row. Sprays were applied at 4 to 5 day intervals and from 50 to 150 gallons per acre. Treatments were started within 5 days after emergence of seedlings and were stopped at approximately the time first fruit were set.

The types of data were recorded. A definite number of leaflets were collected at random from each plot and the leaf spots were counted. Also, disease severity rating based on the percentage of each plant diseased was used as the second criterion of evaluation. The former method was considered more accurate in evaluation of the treatments, while the latter was more useful in indicating disease severity.

EXPERIMENTAL RESULTS

Test No. 1: Seed were planted on June 23. Treatments were evaluated on plants in single row plots, 17 feet long and 7 feet from other plots on the ends and sides. There were four replications.

Bacterial spot lesions were first observed on July 30. The disease increased in severity during a rainy period the first week of August and increased slightly thereafter. The disease developed only to a moderate degree of severity. Ratings were made of the incidence of the disease in each plot (Table 1).

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Table 1. Control of bacterial spot of tomato with certain materials applied as sprays.

Treatment[c]	Concentration per 100 gallons	Average lesions per leaflet[a]	Average disease severity[b]
No treatment	-----	6.5	3.5
Nabac	1.0 lb.	6.1	4.0
Dyrene	2.0 lbs.	5.2	3.5
Streptomycin	100 ppm	1.6	2.3
TBCS	4.0 lbs.	1.2	2.3
TBCS + streptomycin	4.0 lbs. + 100 ppm	0.8	1.0
L.S.D. .05		1.5	
L.S.D. .01		2.0	

[a] Average number of lesions per leaflet on 25 leaflets per plot on August 13. Average of four replicates.

b Disease severity ratings were based on percent of plant affected using a 0 - 10 scale where 0 indicates no disease. Data recorded August 26.

c Active ingredients were Nabac, 12% 2, 2'-methylene bis (3, 4, 6-trichlorophenol); Dyrene, 50% 2, 4-Dichloro-6-(o-chloroanalino) Triazine; (Source of streptomycin was Agrimycin 100), 15% streptomycin sulfate, 1.5% oxytetracycline; TBCS (Tribasic Copper Sulfate) 53% copper.

Streptomycin, Tri-basic Copper Sulfate (TBCS) and the combination of streptomycin plus TBCS all gave good control of bacterial spot. Based on lesions counted, best control was provided by streptomycin plus TBCS, but differences in the protection provided by streptomycin, TBCS or streptomycin plus TBCS in this test were not great. Dyrene and Nabac were ineffective.

Test No. 2: Compatibilities of fungicides with bactericides were checked in this test. The plots were seeded on August 18. Materials were applied to plants in two-row plots. One row of each plot consisted of the variety Homestead-24 and the other of Indian River. The plots were 25 feet long and separated by a guard row of Homestead plants. Four replications were used. Only the Indian River variety was rated for bacterial spot, since it is resistant to gray leaf-spot, the most serious fungus disease at this time of year. The Homestead variety, which is susceptible to gray leaf-spot, was used to evaluate the fungicides.

Bacterial spot lesions were found on the cotyledons soon after emergence. The disease spread with each rain, but did not develop severely enough to result in serious damage to the plants. Results are reported in Table 2.

As in Test 1, TBCS, streptomycin, and TBCS plus streptomycin provided good protection from bacterial spot. The addition of Dyrene to TBCS or to TBCS plus streptomycin had no visible effect on disease development. Likewise, a weekly, but separate, application of maneb did not alter the control of bacterial spot by TBCS plus streptomycin.

Although plants sprayed with TBCS plus streptomycin seemed to have fewer lesions, differences were small. Plants treated with Dyrene plus streptomycin had slightly more bacterial spot than TBCS plus streptomycin. Since Dyrene had little effect on bacterial spot development, streptomycin was inferior to TBCS plus streptomycin.

Test No. 3: Three new materials and five copper compounds were included in this test. The seed were planted on September 12. Plants in single row plots 17 feet long and 7 feet from other plots on the ends and sides were sprayed. Six replications comprised the test.

The first bacterial spot lesions were found when the plants were in the cotyledon stage. The disease increased but never became severe. Ratings of disease incidence are reported in Table 3.

Of the three new materials tested, only the copper salt of Omadine gave any appreciable control. CM-19 was not effective and Nabac-25 was only slightly better than the untreated check.

The five copper compounds, copper salt of Omadine, 658 Fungicide, COCS, Copper A and TBCS, protected the plants equally well. The addition of streptomycin to TBCS did not give improved control in this test.

Table 2. Control of bacterial spot of tomato with certain materials applied as sprays.

Treatment[c]	Concentration per 100 gallons	Average lesions per leaflet[a]	Average disease severity[b]
No treatment	-----	5.7	3.5
TBCS	4.0 lbs	0.9	2.0
TBCS + streptomycin	4.0 lbs. + 100 ppm	0.1	1.0
TBCS + streptomycin maneb (weekly)[d]	4.0 lbs. + 100 ppm 1.5 lbs.	0.3	1.0
TBCS + streptomycin + Dyrene	4.0 lbs. + 100 ppm + 1.0 lb.	0.1	0.8
TBCS + Dyrene	4.0 lbs. + 2.0 lbs.	1.2	1.5
Streptomycin + Dyrene	100 ppm + 2.0 lbs.	1.9	2.0
L.S.D .05		1.4	
L.S.D .01		1.8	

[a] Average number of lesions per leaflet on 25 leaflets per plot on October 22. Average of four replicates.

[b] Disease severity ratings were based on percent of plant affected using a 0-10 scale where 0 indicates no disease. Data recorded on October 23.

[c] Active ingredients were: TBCS (Tri-basic Copper Sulfate), 53% copper; (Source of streptomycin was Agrimycin 100), 15% streptomycin sulfate, 1.5% oxytetracycline; (Source of maneb was Manzate), 70% maneb; Dyrene, 50% 2,4-Dichloro-6-(o-chloroanalino) Triazine.

[d] Maneb was applied at weekly, but separate applications.

Table 3. Control of bacterial spot of tomato with certain materials applied as sprays.

Treatment[c]	Concentration per 100 gallons	Average lesions per leaflet[a]	Average disease severity[b]
No treatment	-----	6.9	3.7
Nabac-25	0.5 lb.	5.7	3.2
CM-19	2 qts.	7.7	3.8
Copper salt of Omadine	2.0 lbs.	0.5	1.5
658	2.0 lbs.	0.9	1.5
COCS	6.0 lbs.	0.2	1.5
Copper A	4.0 lbs.	0.7	1.2
TBCS	4.0 lbs.	0.9	2.0
TBCS + streptomycin	4.0 lbs. + 100 ppm	0.4	1.3
L.S.D .05		0.8	
L.S.D .01		1.1	

[a] Average number of lesions per leaflet on 20 leaflets per plot on October 21. Average of six replicates.

[b] Disease severity ratings were based on percent of plant affected using a 0-10 scale where 0 indicates no disease. Data taken November 3, 1958.

[c] Active ingredients were: Nabac, 25% 2,2'-methylene bis 3, 4, 6-trichlorophenol); CM-19, 17% Phenylphenols and related aryl phenols, 2% octyl and related alkylphenols; Omadine, 50% copper salt of 2-pyridenethione 1-oxide; 658, 95% copper zinc chromate complex (29.6% copper); COCS, 33% copper; Copper A, 45% copper; TBCS, 54% copper; (Source of streptomycin was Agrimycin 100), 15% streptomycin sulfate, 1.5% oxytetracycline.

Phytotoxicity: Most of the materials used in these experiments were relatively non-phytotoxic, even on the cotyledons of seedlings. However, some did cause injury. Nabac-25 (0.5 pound per 100 gallons) caused damage to very young plants but did not injure larger plants. The Dyrene plus TBCS combination caused a slight leaf crinkle on plants during the high temperature period that existed through September. Thereafter the injury was not observed. Repeated sprays of COCS resulted in a stunting of plants.

DISCUSSION

The outstanding observation in the three experiments reported here was the relatively good control of bacterial spot by the copper materials. In past work copper plus streptomycin has been more effective than copper alone under severe conditions (1, 2, 3, 4). It must be pointed out that conditions for these tests were relatively mild and may not have been severe enough to differentiate between effects of copper alone and copper plus streptomycin. On the other hand, in these experiments copper materials did provide considerable protection under mild conditions. As a result, copper can be recommended to farmers who have plants too large to treat economically with streptomycin.

The new materials CM-19 and Nabac failed to control bacterial spot of tomato. Failure to control this disease even under the mild conditions of these tests eliminates their present formulations for use against bacterial spot. Also, the phytoxicity of Nabac precludes its use during hot weather.

It should be emphasized that not less than three nozzles were used and the seedlings were sprayed within 5 days after emergence. It is felt that everything possible should be done early in the growing season to protect the seedlings. Not only is this necessary to protect the seedlings, but such protection has the effect of delaying an increase of the inoculum potential which seems to be so important in the performance of copper or copper plus streptomycin sprays. Both treatments afford better protection in fields relatively free of bacterial spot than in fields where the disease is well established.

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MANEB INJURY TO TOMATO AND PEPPER SEEDLINGS GROWN UNDER GLASS[1]

Colin D. McKeen

Abstract

Experiments conducted at Harrow, Ontario have shown that it is unsafe to spray tomato and pepper seedlings grown under glass with maneb fungicides. Characteristic injury appears on the hypocotyl as a collar of collapsed epidermal and cortical tissue just above the soil line.

INTRODUCTION

To gain full advantage of the frost-free growing season in Ontario locally-grown tomato plants for field setting are started under glass. Depending on the degree of earliness desired, they are grown in greenhouses for 3 to 8 weeks. During this period fungicides are applied regularly to prevent certain diseases.

Observations at the Research Station at Harrow, and also in commercial establishments, have shown that whereas maneb fungicides are non-phytotoxic when used in the field, they are frequently phytotoxic when applied under glass.

OBSERVATIONS

In April 1955 a plant grower at Harrow suffered serious losses when tomato seedlings in greenhouse beds were sprayed with Manzate. In 1958 another plant grower near Harrow suffered appreciable losses when tomato and pepper seedlings were sprayed with Manzate. Also in 1958, in plant beds near Simcoe, Ontario, tomato seedlings showed serious injury after being sprayed with Dithane M-22. Each of these materials applied only once at the rate of 1 1/2 pounds per 100 gallons of water caused the type of injury shown in Figure 1. Epidermal and cortical tissues of the hypocotyl just above the soil line collapsed and died. Moderately to severely injured seedlings toppled over within 24 hours of spraying. However, few of the collapsed seedlings succumbed, because the thread-like strand of vascular tissue remained functional and kept the upper plant parts turgid. Within 2 or 3 days adventitious roots developed above the injured portion of the hypocotyl and made contact with the soil. The remarkable recovery of many of the injured seedlings notwithstanding, growth of the plants was seriously checked. Transplanting costs were also increased because of the extra care required in handling of affected plants possessing crooked stems.

A second application at the first mentioned site, at the rate of one pound of Manzate per 100 gallons of water, caused less but still appreciable injury. In contrast, field spraying of tomato crops in Ontario with maneb at rates of 3 to 4 pounds has not been observed to be phytotoxic.

When greenhouse-grown tomato seedlings 2 to 3 inches in height were sprayed with maneb fungicides at the Research Station, Harrow, during the months of May, June, July and August, 1955, characteristic symptoms of injury developed (Fig. 1). Somewhat older transplanted seedlings (Fig. 3) were also found to be susceptible to injury. Injury on the hypocotyl or stem consistently showed as a collar of collapsed epidermal and cortical tissue just above the soil line. Small chlorotic to necrotic spots developed infrequently on the cotyledons and leaves.

Pepper (Capsicum frutescens) seedlings show injury similar to that of tomatoes (Fig. 2). No injury has been observed on tomatoes or peppers sprayed after plants are 7 or 8 weeks old, at which time stems show considerable hardening and thickening.

Injury has been most severe under crowded conditions of plant growth. In stands of tomato and pepper seedlings, containing four or more seedlings per square inch, a carpet of fairly dense foliage develops at 12 to 15 days after emergence. Under these conditions hypocotyl tissue remains relatively "soft." After the spray has been applied, fungicidal droplets tend to collect along the hypocotyls most frequently just above the soil line, and it is several hours before they evaporate. Most phytotoxicity due to maneb occurs near the base of the hypocotyls.

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[1]Contribution No. 5, from the Research Station, Research Branch, Canada Department of Agriculture, Harrow, Ontario.

FIGURE 1. Maneb injury to 10-day-old tomato seedlings. Note the collar of injured tissue on hypocotyl just above the primary root.

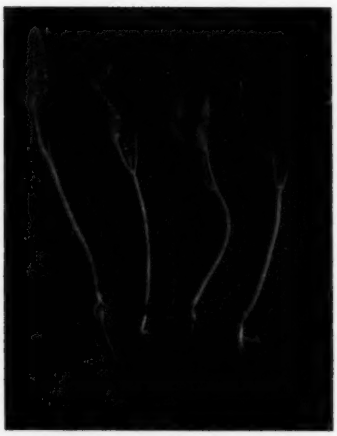

FIGURE 2. Maneb injury to 12-day-old pepper (California Wonder) seedlings. Note some necrotic spots on cotyledons in addition to hypocotyl injury.

FIGURE 3. Maneb injury on 20-day-old tomato plants. At transplanting, the hypocotyls were covered with soil up to the cotyledons.

Injury has occurred regardless of the time of day that plants are sprayed. Little difference in the extent of injury was noted when sprays were applied during either sunny or cloudy weather.

Results of experiments conducted in 1956 failed to indicate that "aging" of Manzate increased its phytotoxicity. As a matter of fact injury caused by one lot of the material which had been stored in the laboratory for 3 years was less extensive than that resulting from the use of a newly acquired lot of the same formulation. It is not presently known whether the conditions of fungicidal storage might alter the phytotoxic properties of the material.

Only dithiocarbamate fungicides containing manganese have been found to cause the above-mentioned injury.

DISCUSSION

The chemical decomposition of nabam and its interaction with heavy metal salts has been worked out by Ludwig et al. (3), and Klopping and van der Kerk (1). Ludwig et al. (3) concluded that the formation of a heavy metal salt of nabam increases the amount of spray deposit, stabilizes it on the foliage thus permitting a longer oxidation period, and reduces losses through the deleterious acid breakdown. In more recent studies, Ludwig and Thorn (2) state that "the mechanisms of reaction between nabam and a metal such as manganese are not easy to resolve." They suggest, however, that manganese has a catalytic effect on the oxidation process of nabam. While it was pointed out above that the high incidence of injury to tomato and pepper seedlings occurred in close proximity to the soil, and often appears to be associated with the slow evaporation of spray droplets, it may also be true that conditions at this locus favor the deleterious acid decomposition of maneb.

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VEGETABLE DISEASE CONTROL IN DELAWARE IN 1958[1]

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Vegetable disease research in Delaware in 1958 was designed to compare several promising new fungicides with currently recommended materials for control of a variety of diseases. The crops and diseases concerned were: (a) tomato -- anthracnose (Colletotrichum phomoides) and gray leaf spot (Stemphylium solani); (b) pepper -- ripe rot (Colletotrichum capsici); (c) cantaloupe -- leaf spot (Alternaria cucumerina); and, (d) lima bean -- stem anthracnose (Colletotrichum truncatum). All foliar sprays were applied with a four-nozzle boom with drop nozzles designed to apply materials to both upper and lower leaf surfaces at 300 psi. Experimental plots were located at the University Farm, Newark, Delaware, or in fields on various farms in the State. A randomized, replicated block design was used in all cases. Pertinent data for each test are presented in tabular form under each crop.

TOMATO (LYCOPERSICUM ESCULENTUM, VARIETY V. R. MOSCOW)

The fungicides Dithane M-22, Phaltan, and Dyrene were compared with untreated rows in a randomized block design of 12 replications. Tomato anthracnose was serious at the plot site in 1957. Stemphylium leaf spot was induced to occur by inoculating the end plant of each row with a spore and mycelial suspension of the fungus 1 week prior to the first application of fungicides. Fungicidal sprays were applied eight times between July 7 and August 26 at from 5 to 8 day intervals, depending on rainfall which was heavy and well distributed. The fruit were harvested five times and graded as to marketable, number 2-anthracnose or cull-anthracnose. The percentage of foliage lost due to leaf spot was determined by the index method of evaluation and the results averaged; the data obtained are presented in Table 1.

Table 1. Control of anthracnose and Stemphylium leaf spot of tomato, Newark, Delaware, 1958.

Treatment[a]	Pounds/acre	Number anthracnose cull fruit[b]	Percentage defoliation : Aug. 15	Sept. 9
Untreated	---	119	53	68
Dithane M-22	3	25	12	12
Phaltan	3	20	15	42
Dyrene	4	35	13	12
L.S.D. (.05)		13		

[a]8 applications July 7-August 28, 1958; Dithane M-22, Rohm & Haas Co.; Phaltan, California Spray Co.; Dyrene, Chemagro Co.
[b]Average of 12 replicates, 12 plants per replicate.

The data indicate that, under the conditions of this test, the fungicide Phaltan was as good as Dithane M-22 (maneb) for anthracnose control but much poorer for gray leaf spot control, particularly as the season progressed. Dyrene had a higher number of fruit infected with anthracnose than did maneb, but this was not statistically significant; both Dyrene and Dithane M-22 (maneb) gave excellent control of gray leaf spot. Phaltan was significantly better than Dyrene for anthracnose control but inferior for gray leaf spot control. All compounds resulted in excellent anthracnose and leaf spot control compared with unsprayed rows.

PEPPER (CAPSICUM ANNUUM, VARIETY YOLO WONDER)

A field site was chosen on a Delaware farm that had a previous record of disease loss due to ripe-rot (Colletotrichum capsici). The plot consisted of three treatments replicated four

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times, using 25 feet of row per replicate, at the edge of a commercial field of peppers. Six spray applications were made, starting at first fruit set and continuing until the fruit began to turn red. The fruit were harvested when the majority of fruit were in the red-ripe stage and scored as to number of marketable fruit, number 2-anthracnose, and cull-anthracnose. The results are presented in Table 2.

Table 2. Control of pepper ripe-rot, Marydel, Delaware, 1958.

Treatment[a]	Pounds/acre	Average number infected fruit/replicate[b]
Untreated	---	45.6
Dithane Z-78	3	14.0
Manzate	3	8.4
L.S.D. (.05)		23.9

[a]Six sprays July 9-August 26, 1958 at average interval of 9 days; Manzate, duPont Co., Dithane Z-78, Rohm and Haas Co.
[b]Average of four replicates; combined categories of number 2- and cull-anthracnose.

The disease occurred naturally and caused approximately a 30 percent loss in this 7-acre field of peppers. The application of fungicides in the experimental plot area materially reduced the incidence of disease. Neither fungicide was statistically superior to the other, but both gave significant control compared with untreated rows.

CANTALOUPE (CUCUMIS MELO, VARIETY HALES BEST)

A randomized block design of three replications was used to evaluate four fungicides for the control of Alternaria leaf spot of cantaloupe. Each treatment row was 30 feet long. The percentage of foliage lost was estimated by the index method. The materials used and results obtained are presented in Table 3.

Table 3. Control of Alternaria leaf spot of cantaloupe, Newark, Delaware, 1958.

Treatment[a]	Pounds/acre	Percentage defoliation September 15, 1958[b]
Untreated	---	83
Dithane Z-78	3	30
Phaltan	3	27
Dyrene	4	14
Dithane M-22	3	13

[a]Six applications July 28-September 10, 1958.
[b]Average of three replications.

By the end of August leaf spot was severe in the plot. All fungicides gave control of leaf spot, but Dyrene and Dithane M-22 were outstanding. None of the materials caused discernible phytotoxicity.

LIMA BEAN (PHASEOLUS LIMENSIS, VAR. THOROGREEN)

Data was obtained on the control of stem anthracnose (Colletotrichum truncatum) in a plot located in a commercial planting of lima beans at Clayton, Delaware. This disease is one of the causes of pod and seed blemish and accounts for part of the problem encountered in canneries under the general name of "seed blotch" and "soil rot." The symptoms on the pods are similar to those caused by Rhizoctonia solani.

A randomized block design of four replications was used to evaluate Dithane M-22, Phaltan, Orthocide 80W, Dyrene, and Tribasic Copper Sulfate for disease control. Each

treatment row was 100 feet in length. Four sprays were applied at weekly intervals, beginning when the first pods set were about 3 inches in length. About 2 weeks after the last spray, 50 plants were pulled at random from each treatment row and the marketable-sized pods removed and graded into infected and non-infected categories. The materials, concentration used, and results are presented in Table 4.

Table 4. Control of stem anthracnose of lima bean, Clayton, Delaware[a].

Treatment[b]	Pounds/acre	Average number infected pods[c]	Percent of total
Untreated	---	69.0	7.0
Dithane M-22	3	7.5	0.8
Phaltan	3	33.3	3.3
Ortho 80W	1 1/2	44.3	4.3
Dyrene	4	21.5	1.7
Tribasic $CuSO_4$	4	40.0	3.7
L.S.D. (.05)		16.4	

[a]Four sprays: September 4, 11, 18 and 24.
[b]Ortho 80W, California Spray Co.; Tribasic Copper Sulfate, Tennessee Copper Corporation; (See Table 1 for others).
[c]Average of four replications.

Dithane M-22 and Dyrene were significantly better for control of stem anthracnose than Phaltan, Ortho 80W, or Tribasic Copper Sulfate. The latter-named materials were not significantly different in degree of control obtained. All materials gave significant control compared with untreated rows.

Based on the results presented above on the control of five different fungal pathogens on four different host plants, it is readily apparent that maneb (Dithane M-22; Manzate) is an outstanding fungicide for control of a wide range of vegetable diseases.

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CONTROL OF DOWNY MILDEW OF BROCCOLI WITH FUNGICIDES AND FUNGICIDE-STREPTOMYCIN COMBINATION SPRAYS[1,2]

John J. Natti

Summary

Copper-Zinc, Copper-Zinc plus streptomycin, maneb plus streptomycin, and Agrimycin 500 were the most effective of the materials tested in the field for the control of downy mildew of broccoli. The combination of Copper-Zinc plus streptomycin, although only slightly more effective than Copper-Zinc alone during the time of spray applications, was far more effective than Copper-Zinc alone 21 days after the final spray application. Maneb plus streptomycin, although equally as effective as Copper-Zinc as a protective spray during the period of spray applications, did not provide control after the discontinuance of spray applications. The incidence of infected broccoli heads was less in plots sprayed prior to the development of broccoli heads than in plots sprayed only during the period of head development.

Downy mildew, caused by Peronospora parasitica Pers. ex Fr., is an important disease of broccoli and other crucifers in many areas of the United States. The only method of control at the present time is the application of protective foliar fungicides. Control by fungicide applications, however, has not been as effective as desired, mainly because of inadequate coverage of the foliage with the fungicide during rainy periods. Because rainy conditions are favorable for the spread and germination of the downy mildew spores and the rains also wash spray deposits from the broccoli leaves, the times at which control is most necessary are also the times at which the plants are the least protected. To be effective for control of downy mildew of broccoli, therefore, a fungicide must either be retained at fungitoxic concentrations on the foliage during rains or it must be absorbed by the leaves so that it is not subjected to weathering. Field spray tests were conducted in 1957 and 1958 to determine whether certain fungicides and fungicide-streptomycin combinations possessed the property of tenacity on foliage or of systemic activity essential for effective control of downy mildew of broccoli.

MATERIALS AND METHODS

The plot design, method of spray application, and the method of obtaining data on control of downy mildew were essentially the same in 1957 and 1958. Broccoli plants of the variety Waltham 29 were used in all tests. The plants were started in a seedbed during the first week of June and were transplanted in the test plots on July 17 and 18. Each spray plot consisted of two adjacent 25-plant rows. A single unsprayed row separated the spray plots. In the 1957 tests, each treatment was replicated five times and in the 1958 tests, four times. The sprayer consisted of a hand-operated spray rig equipped with a boom and drop-pipes in such a manner that the sprays were delivered from one overhead nozzle and from one nozzle 6 inches above the soil level on either side of the plants. The sprays were applied at the desired concentration in 90 gallons per acre at an air pressure of 45 psi. Compressed CO_2 was used to discharge the spray.

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[1]Approved by the Director as Journal Paper No. 1162, New York State Agricultural Experiment Station, Geneva, New York.

[2]The cooperation of the following companies who supplied test materials is acknowledged: American Cyanamid Company for Cyprex and AC 23441; California Spray-Chemicals Corporation for Phaltan 50W and Orthocide 50W; Chas. Pfizer and Company, Inc. for Agrimycin 100 and for various formulations of Agrimycin 500; E. I. duPont de Nemours and Company for Manzate; Gallowhur Chemical Corp. for G 1143; Merck and Company, Inc. for Agri-Strep; Naugatuck Division of U. S. Rubber Company for Spergon; Olin-Mathieson Chemical Corporation for Omadine salts; and Tennessee Corp. for Copper-Zinc.

Data on control of downy mildew on the foliage were obtained by counting the number of lesions in 9 square inches of leaf area per leaf on 25 leaves from each plot. Data on incidence of systemic infection of the broccoli heads were obtained by examining the interior of 25 heads from each plot.

The 1957 and 1958 tests differed as indicated by the following procedures:

1957 Tests

Sprays were applied on August 20, 27, September 4, 11, 17, and 24. The plots were scored for foliage infection on September 23 to obtain data on control during the period of spray applications. The plots were again scored on October 15, twenty-two days after the final spray application, to obtain data, as indicated by disease control, on the tenacity of the spray materials on the foliage and on possible systemic activity.

1958 Tests

Tests were conducted at two locations. At Location I, sprays were applied on August 27, September 4, 11, and 23. The original plan of this test was to apply six sprays at about weekly intervals. Heavy rains forced the postponement of the fourth spray application for 5 days. The spread of downy mildew and the rank growth of the plants between the third and fourth spray applications made further spray applications impractical. Data on control of foliage infection and on incidence of head infection were taken on September 29.

At Location II, in addition to evaluating the spray materials for control of downy mildew, the test was designed to obtain information on the stage of development of the broccoli plant at the time of initiation of systemic infection of the head. Three different spray schedules were used. In the first schedule (three early sprays), sprays were applied on August 26, September 3 and 11. The plants sprayed according to this schedule were protected up to about 1 week of the beginning of head development. In the second schedule (six all season), sprays were applied on August 26, September 3, 11, 18, 23, and 30 to protect the plants prior to and after head development. In the third schedule (three late sprays), sprays were applied on September 18, 23 and 30. The plants in these plots were protected only during the period of development of the heads to marketable maturity. Data on foliage infection and systemic infection of the heads were obtained on October 1 and 2.

RESULTS

1957 Test

Downy mildew was more severe early in the season than later. In the seedbed, the lower leaves of most of the seedlings were severely infected. Soon after the plants were transplanted to the spray plots, the infected leaves abscised and the new growth remained free from mildew until the first week of September. On September 23, the incidence of downy mildew was significantly less in all sprayed plots than in the unsprayed (Table 1). Manzate at 3 pounds plus Agri-Strep at 1 pound, Copper-Zinc at 6 pounds, copper salt of Omadine at 6 pounds, Manzate at 6 pounds, and Agri-Strep at 1 pound provided comparable control and were more effective than the other treatments.

On October 15, twenty-two days after the final spray application, Copper-Zinc, Manzate plus Agri-Strep, and Agri-Strep were by far the most effective of the treatments (Table 1). Control provided by the combination of Manzate plus Agri-Strep appeared to be due entirely to Agri-Strep. The effectiveness of Copper-Zinc can be attributed to its tenacity, since foliage sprayed with this material still retained a noticeable spray deposit. Control provided by Agri-Strep appeared to be of systemic nature. On October 16, twenty-three days after the last spray application, washings from leaves sprayed with Copper-Zinc inhibited the germination of mildew spores in test tubes, whereas washings from leaves sprayed with Agri-Strep had no effect on the germination of the spores.

Small brown sunken spots occurred in scattered areas in leaves sprayed with Copper-Zinc and with the copper salt of Omadine. This injury did not appear to have any effect on the growth of the plants. Cyprex and AC 23441 caused a pronounced yellowing of the foliage, necrosis of the margins of the older leaves, and stunting of the plants. No bleaching of the foliage of plants sprayed with Agri-Strep was observed.

Table 1. Control of downy mildew of broccoli, field spray test, 1957.

Treatment[a]	Pounds per acre[b]	Lesions per leaf[c] 9/23	Lesions per leaf[c] 10/15	Injury rating[d]
Manzate + Agri-Strep	3+1	4	8	0
Copper-Zinc	6	4	10	+
Agri-Strep	1	5	11	0
Copper Omadine	6	5	18	+
Manzate	6	8	21	0
G-1143	6	12	21	0
Cyprex	3	12	22	+++
Spergon SL	4	13	22	0
Phaltan 50W	6	15	23	0
AC 23441	3	11	24	+++
Manzate	4	12	25	0
Zinc Omadine	6	18	31	0
None	-	24	34	0
LSD 19:1		4	4	
99:1		5	5	

[a]Plots sprayed on August 20, 27; September 4, 11, 17 and 24.
[b]Pounds of formulation in 90 gallons per acre.
[c]Average number of lesions in 9 square inches of leaf area per leaf from 25 leaves from each of five replications.
[d]Injury rating: + = slight, ++ = moderate, +++ = severe.

Table 2. Control of downy mildew of broccoli, field spray test, Location I, 1958.

Treatment	Pounds per acre[a]	Number of lesions[b]	Injury rating[c]
Spergon	6.0	21	+
Manzate	6.0	17	+
Phaltan 50W	6.0	22	+
Phaltan 50W + ML-107[d]	6.0	18	++
Orthocide 50W	6.0	31	
Orthocide 50W + ML-107[d]	6.0	25	+
Copper-Zinc	6.0	10	+
Agrimycin 500 (a)[e]	4.6	9	+
Agrimycin 500 (b)[f]	4.6	10	++
Agrimycin 500 (c)[g]	4.6	7	+++
Agrimycin 100[h]	0.5	20	+
Agrimycin 100[h]	1.0	11	++
None	-	36	
LSD 19:1		2.5	
99:1		3.4	

[a]Pounds of formulation in 90 gallons per acre.
[b]Average number of lesions in 9 square inches of leaf area per leaf from 25 leaves from each of four replications.
[c]Injury rating: + = slight, ++ = moderate, +++ = severe.
[d]ML-107 used at rate of 675 ml in 90 gallons.
[e]Agrimycin 500 (a) contains 42.4% copper and 1.76% streptomycin.
[f]Agrimycin 500 (b) contains 35.3% copper and 3.0% streptomycin.
[g]Agrimycin 500 (c) contains 10% copper and 10% streptomycin.
[h]Agrimycin 100 contains 15% streptomycin and 1.5% oxytetracycline.

Table 3. Control of downy mildew of broccoli, field spray test, Location II, 1958.

Treatment	Pounds per acre[a]	Number of spray applications[b]	Number of lesions[c]	Incidence of infected heads[d] (percent)
Manzate	4.0	6 (all season)	22	2
	4.0	3 (early)	35	3
	4.0	3 (late)	35	4
Manzate + Agri-Strep	4.0 + 0.4	6 (all season)	8	0
	4.0 + 0.4	3 (early)	30	0
	4.0 + 0.4	3 (late)	34	5
Agri-Strep[e]	0.4	6 (all season)	27	0
	0.4	3 (early)	34	0
	0.4	3 (late)	37	3
Copper-Zinc	4.0	6 (all season)	10	0
	4.0	3 (early)	21	0
	4.0	3 (late)	28	6
Copper-Zinc + Agri-Strep	4.0 + 0.4	6 (all season)	7	0
	4.0 + 0.4	3 (early)	13	0
	4.0 + 0.4	3 (late)	31	4
Agrimycin 500 (a)[f]	4.6	6 (all season)	11	0
	4.6	3 (early)	16	0
	4.6	3 (late)	24	3
Spergon	4.0	6 (all season)	25	2
None	-	0	49	8
LSD[g] 19:1			3	
99:1			4	

[a]Concentration of formulation in 90 gallons per acre.
[b]6 (all season) = 6 sprays applied on August 26, September 3, 11, 18, 23, and 30.
3 (early) = 3 sprays applied on August 26, September 3, and 11.
3 (late) = 3 sprays applied on September 18, 23, and 30.
[c]Average number of lesions in 9 square inches of leaf area per leaf from 25 leaves from each of four replications.
[d]Percentage based on 25 heads examined from each of four replications.
[e]Agri-Strep contains 37% streptomycin sulfate; 0.4 pounds in 90 gallons contains active streptomycin at concentration of approximately 200 ppm.
[f]Agrimycin 500 (a) contains 42.4% copper and 1.76% streptomycin; concentration of streptomycin in 90 gallons is 108 ppm.
[g]Data on three late sprays not included in statistical analysis.

1958 Tests

The frequent rains that occurred during the growing season provided the opportunity to compare the test materials under rigorous disease control conditions. At Location I, Copper-Zinc was the most effective of the treatments with fungicide alone (Table 2). Manzate, which ranked second, was slightly more effective than Spergon and Phaltan 50W. Orthocide 50W was the least effective. The sticker ML-107 increased to a slight extent the control provided by Phaltan 50W and Orthocide 50W.

The Agrimycin 500 formulations, each of which contain tribasic copper sulfate plus streptomycin and oxytetracycline, and Agrimycin 100, which contains only the antibiotics, gave control comparable to that provided by Copper-Zinc alone. Under the conditions of this test, no evidence of additive or synergistic activity from the combination treatments was observed.

No data on incidence of infected heads were taken in this test.

At Location I, all of the materials with the exception of Orthocide 50W caused some injury, probably because of the succulent condition of the plants. The formulations of Agrimycin 500 which contained the higher concentrations of streptomycin caused moderate to severe bleaching of the foliage (Table 2).

At Location II, control of downy mildew in plots sprayed according to the 6-spray appli-

cation schedule indicates the comparative effectiveness of the test materials during the period of spray applications. Under the conditions of this test, Copper-Zinc was definitely superior to Manzate, Spergon, and Agri-Strep (Table 3). The combination of Manzate and Agri-Strep was about three times more effective than either material alone and equalled Copper-Zinc in performance. The combination of Copper-Zinc and Agri-Strep was slightly more effective than treatments with Copper-Zinc alone. Agrimycin 500, a commercial formulation containing tribasic copper sulfate plus streptomycin and oxytetracycline, was slightly less effective than the Copper-Zinc plus streptomycin combination.

At Location II, control of downy mildew in plots sprayed according to the 3-early spray schedule indicates the residual or possible systemic activity of the test materials. These plots were scored 21 days after the final spray application. Copper-Zinc plus Agri-Strep, and Agrimycin 500 were the most effective of the treatments (Table 3). The combination of Manzate plus Agri-Strep, although effective as a protective spray during the time of spray applications, did not provide effective control after the sprays were discontinued. Agri-Strep alone provided only mediocre control. Copper-Zinc exhibited considerable residual activity.

Data on foliage infection in plots sprayed according to the 3-late spray schedule were not included in the statistical analysis on the performance of the test materials because most of the infection in these plots occurred before any sprays were applied. The relatively high incidence of mildew in these plots indicated that none of the test materials possessed eradicative properties.

The incidence of infected heads was low, even in the unsprayed plots (Table 3). Most of the infected heads were present in plots that received sprays only during the period at which the heads were developing into marketable maturity. No infected heads were present in plots sprayed with Copper-Zinc or with Agri-Strep prior to head development. These results suggest that systemic infection of the heads is initiated before the heads are formed and that spray applications made after the heads begin to form are too late to be of value in protecting them against infection.

DISCUSSION

Copper-Zinc is superior to Manzate and Spergon for the control of downy mildew of broccoli. The superiority of Copper-Zinc depends to a great extent upon its tenacity to the foliage. This tenacity is reflected in control during rainy periods and over an extended period after cessation of spray applications. Copper causes slight injury to broccoli foliage but this injury does not appear to affect yields.

Streptomycin provides control by both surface protective and localized systemic activity (9). However, it appears that effective control with streptomycin cannot be obtained at non-phytotoxic levels of the antibiotic. The combination of streptomycin and fixed copper offers considerable promise for the control of downy mildew as indicated by the performance of Agri-Strep plus Copper-Zinc and of Agrimycin 500 in these tests. The systemic activity of streptomycin and the tenacity of the copper to broccoli foliage make this combination very effective against mildew even under severe weathering conditions. It is possible that the concentrations of both copper and streptomycin can be reduced in the combination sprays so that the danger of injury to the foliage is eliminated entirely.

The mechanism involved in the increased effectiveness of the combination sprays is not known, but some indications as to its nature can be derived from published reports on the control of various diseases by combination treatments. Copper-streptomycin combination sprays have been observed to be more effective than either component for the control of bacterial spot of pepper caused by Xanthomonas vesicatoria (Doidge) Dowson (6, 7), bacterial blight of celery caused by Pseudomonas apii Jagger (5), downy mildew of cucurbits caused by Pseudoperonospora cubensis (Berk. & Curt.) Rostow. (4), downy mildew of lima bean caused by Phytophthora phaseoli Thaxt. (11), late blight of potato caused by Phytophthora infestans (Mont.) D By. (1), and ripe rot of pepper caused by Vermicularia capsici Snyd. (3). Bonde and Johnson (1) observed that the increase of effectiveness of fungicide-streptomycin combinations for the control of late blight of potato was not limited to copper-containing fungicides. They also observed that bichloride of mercury, hydroxymercurinitrophenol (Semesan Bel), and captan gave more effective control of potato bacterial ring rot, caused by Corynebacterium sepedonicum (Spieck. & Kotth.) Skapt. & Burkh., when combined with streptomycin than when used alone (2). In view of the above observations, the increased effectiveness of fungicide-streptomycin, or of bactericide-streptomycin combinations, is not specific for any given pathogen, suscept, fungicide, or bactericide. The nonspecificity of the fungicide or bactericide

component in increasing the effectiveness of the combination treatments may be considered as evidence that streptomycin is the initial activator of the combination. Vörös et al. (10) suggest that streptomycin does not exert any direct effect on the pathogen but provides protection by stimulating the polyphenolase activity of the plant so that polyphenol derivatives in fungitoxic concentrations are produced in the plant. They also suggest that since polyphenolases are copper enzymes, one of the roles of copper in copper-streptomycin combinations is to supply copper to the plant so that the increased polyphenolase activity may be maintained.

The mechanism of the increased effectiveness of the pesticide-streptomycin combinations over that of either component alone has been referred to as synergism (1, 11), supplementary synergism (5), and additive effect (2, 3, 5, 7, 11). The mechanism suggested above by Vörös et al. (10) would probably have to be classed as a form of potentiated synergism according to Horsfall's (8) definitions of types of synergism. A better knowledge of the mechanism of the increased effectiveness of the combination treatments would not only serve to clarify its definition, but also would very probably reveal some important fundamental aspects on the pesticidal action of combination treatments.

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EVALUATION OF CHEMICAL DIPS FOR CONTROL OF DECAY IN PACKAGED CARROTS[1]

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Abstract

Dipping topped carrots in a 0.01 percent aqueous solution of sodium hypochlorite was generally effective in reducing all forms of decay in packaged carrots stored at 70° and 80° F. Carrots stored at 40° or 50° showed no effect from hypochlorite treatment. In one of the five experiments sodium hypochlorite was sufficiently phytotoxic to increase bacterial and Penicillium decays.

Sorbic acid reduced decay due to Alternaria in only one test and did not affect soft-rot bacteria, Mucor or Penicillium sp.

Bacteria and Mucor sp. were more commonly associated with decay than were Alternaria sp. or Penicillium sp.

There was considerably less decay in carrots held at 40° than in others held at 50° or higher.

MATERIALS AND METHODS

Irrigated Imperator carrots with tops removed in the field were washed at a packing shed preparatory to packaging. Samples of the carrots were immediately taken to the laboratory where they were gently stirred in an aqueous inoculum. The roots were then submerged in one of the disinfectants being studied or in a tapwater control for several minutes, drained and packaged in perforated polyethylene bags, five per bag. The carrots were stored in controlled-temperature, air-circulated, dark rooms. Droplets of condensed moisture in the bags showed that storage was begun in high relative humidity.

Each bag was rated for decay after storage for 7, 14 and 21 days. Severity of each type of decay was indicated by use of ratings: 0 -- none, 1 -- less than 20 percent, 2 -- 20 to 40 percent, 3 -- 40 to 60 percent, 4 -- 60 to 80 percent, 5 -- 80 to 100 percent. Decay ratings were compared by analysis of variance.

Inoculum for the first experiment was prepared by filtering through cheesecloth separate nutrient-broth cultures of soft-rot bacteria, Alternaria sp. and Mucor sp. into water. The cultures had been prepared from isolates obtained from decayed carrot roots.

Sorbic acid (2,4-hexadienoic acid) has been shown to be fungistatic (7, 8) and less toxic than sodium benzoate (3) or the copper materials used by Dye (5). Emard and Vaughn (6) reported an inhibition of fungi by 0.1 percent sorbic acid. Sodium hypochlorite has controlled mold in apples (1) and cantaloupes (2).

In the first experiment, carrots were treated with 0.05 percent, 0.1 percent and a saturated aqueous (approximately 0.15 percent) solution of sorbic acid. Sodium hypochlorite was used at concentrations of 0.01, 0.02 and 0.04 percent aqueous solutions. The carrots were then held at 70° F.

Inoculum for the second experiment was made by stirring decayed carrots from the first experiment in water and filtering as before. By this method, a Penicillium sp. was added to the bacteria, Alternaria, and Mucor spp. The same disinfectants were used as in the first experiment except that 0.15 percent sorbic acid was omitted. The carrots were again held at 70°

In the third experiment, 0.01 and 0.005 percent sodium hypochlorite dips were compared with the control (tap-water) dip, each being used for a 1-minute and a 3-minute immersion. Decayed carrots were again used for inoculum and the packaged carrots were held at 70°

Sodium hypochlorite at concentrations of 0.01, 0.1 and 1.0 percent was used in a fourth experiment and decayed carrots were again used for inoculum. The packaged roots were held at 70°, as before.

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Storage temperatures of 40°, 50°, 70° and 80° were used in a similar fifth experiment to note any relationship of temperature to the effectiveness of a 0.01 percent sodium hypochlorite dip.

The basic statistical analysis compared the variability in average scores for the several treatments with the average variability among bags treated alike, as in a completely randomized experiment.

Source	d.f.	MS		
Among treatments	t-1	TMS	$F = \frac{TMS}{EMS}$	$s_{\bar{x}} = \sqrt{\frac{EMS}{r}}$
Among bags in treatments	t(r-1)	EMS		

The standard error of a treatment mean was calculated for each analysis and the Multiple Range Test (4) was used to separate treatment means where the calculated F exceeded the Tabular F at the 5 percent level. Analyses were made for ratings on each decay due to each organism isolated for the several dates of the test and for ratings of total decay. The means and respective standard errors are presented in Table 1.

RESULTS

Test 1

Analyses showed that decay generally increased significantly with increasing length of storage period.

Sorbic acid caused no reduction in bacterial or Mucor decay, but did significantly reduce damage due to Alternaria. Decay by each organism increased significantly with increasing levels of sodium hypochlorite. However, sodium hypochlorite at concentrations of 0.01 and 0.02 percent was generally effective in significantly reducing decay compared with the control treatment.

Test 2

Significantly more decay was noted after 14 days of storage than after 7 days.

Sorbic acid was ineffective in controlling decay while sodium hypochlorite was effective, with no observable difference between levels of the hypochlorite treatment.

Test 3

No appreciable decay was present in the carrots; hence, at the end of the 7-day storage period evaluation of the treatments was not possible. While some decay due to Mucor and Penicillium was present after 14 days in storage, no effect of treatment was detected.

Test 4

Carrots in this experiment appeared to be injured by sodium hypochlorite. Small scars which are normally found on carrots were whitened by the chemical. Eventually these bleached areas became sunken and black, with no signs of fungus infection.

This physiological injury was significantly increased for all storage groups owing to the presence of sodium hypochlorite. Decay was minimal for the 7- and 14-day groups. After 21 days in storage bacterial and Penicillium decays were significantly greater for treated carrots than controls.

Test 5

A significant reduction in decay for carrots treated with 0.01 percent sodium hypochlorite was observed in all samples held at 80° F. No differences could be detected at 50° or at 40°. Mean ratings for decay were lower for treated than for control carrots held at 70°, but the variation among bags treated alike was too large to detect this difference with a 5 percent chance of being in error.

Table 1. Mean ratings[a] and standard errors for decay by selected organisms, by date and test, for various treatments, based on averages of four bags of five carrots each.

Test 1 -- Held at 70° F

Treatment[b]	7 day Bact.	Mucor	Alter.	Total[c]	14 day Bact.	Mucor	Alter.	Total	21 day Bact.	Mucor	Alter.	Pen.	Total
.05 s	1.50	.00	1.50	2.75	1.75	2.50	2.25	5.00	3.25	5.75	4.00	2.00	9.00
.10 s	1.75	.50	.75	3.00	5.75	5.50	1.00	9.50	4.00	10.00	3.00	1.75	11.00
.20 s	1.75	1.00	.25	2.75	2.25	9.25	1.00	11.00	.75	12.50	.25	4.75	13.50
.01 Cl	.25	.00	.00	.25	1.00	.00	.25	1.00	.50	.50	.25	.25	1.25
.02 Cl	.50	.25	.25	.75	1.25	.50	.50	2.00	1.00	1.25	.25	.50	3.00
.04 Cl	3.25	.75	.25	3.75	7.00	1.25	2.25	8.75	6.75	2.75	.25	2.25	10.25
H_2O	1.25	.75	3.00	5.00	3.00	2.75	4.75	9.75	2.50	4.25	4.75	2.75	11.00
$s_{\bar{x}}$[d]	(NS)	(NS)	.44	.84	1.55	1.32	.44	1.99	(NS)	1.92	1.07	(NS)	2.24

Test 2 -- Held at 70° F

Treatment	7 day Bact.	Mucor	Pen.	Total	14 day Alter.	Mucor	Pen.	Total
.05 s	3.25	8.25	.50	8.50	.00	15.00	13.75	24.00
.10 s	4.75	9.75	2.25	11.25	.25	16.25	15.25	23.75
.01 Cl	1.25	4.00	.25	5.00	.00	9.00	12.25	17.25
.02 Cl	2.25	2.85	1.50	5.50	.00	8.25	13.75	17.75
.04 Cl	.50	4.25	2.50	5.25	.00	9.50	10.50	16.50
H_2O	3.25	6.25	2.75	8.00	.00	13.75	14.75	21.50
$s_{\bar{x}}$	.63	1.08	.59	.92	(NS)	1.66	(NS)	(1.43)

Test 4 -- Held at 70° F

Treatment	7 day Phys. Inj.	Bact.	Pen.	Total	14 day Phys. Inj.	Bact.	Alter.	Pen.	Total	21 day Phys. Inj.	Bact.	Alter.	Pen.	Total
H_2O	1.25	.00	.75	.75	2.50	.00	.00	1.75	1.75	2.25	1.75	1.25	2.00	4.25
.01 Cl	8.75	.00	2.00	2.25	15.25	.00	.25	3.00	3.00	18.00	.25	.25	10.50	11.50
.10 Cl	5.75	.00	1.50	1.50	12.25	.00	.00	3.25	3.25	14.00	1.50	.75	9.00	10.25
1.00 Cl	10.00	.25	3.50	3.75	11.00	1.50	.25	5.25	6.75	15.00	6.25	.50	13.00	15.75
$s_{\bar{x}}$	.98	(NS)	(NS)	(NS)	(1.25)	(NS)	(NS)	(NS)	(NS)	1.61	.81	(NS)	1.55	1.39

Test 5 (Total Decay) Average of Ten Weekly Readings

	80°	70°	50°	40°
H_2O	17.4	11.3	2.9	1.2
.01 Cl	11.4	6.9	3.9	.2
$s_{\bar{x}}$	.68	(NS)	(NS)	(NS)

[a] = 0-none, 1-less than 20 percent, 2-20 to 40 percent, 3-40 to 60 percent, 4-60 to 80 percent, 5-80 to 100 percent decayed.

[b] = The treatment concentrations are percent aqueous solutions of sorbic acid (s) and sodium hypochlorite (Cl)

[c] = Total decay may be less than total of all rots because of some overlap on the samples.

[d] = $s_{\bar{x}}$ is the standard error of a treatment mean based on the pooled error in the analysis of variance. NS = no statistical significance.

After being held at 70° and 80° for a week many packages of carrots in all treatments were in poor condition because of decay. Some of those held at 50° showed a little decay during the third week in storage. None of the carrots held at 40° were decayed until the tenth week of storage.

DISCUSSION

The fact that decay organisms were limited but not eradicated by the chemical dips points out the importance of considering the phytotoxicity of a dipping solution. In the fourth experiment decay eventually became more severe in treated carrots than in the controls. Apparently the ability of this particular batch of roots to ward off infection was diminished by the toxic action of sodium hypochlorite.

Since dipping carrots in 0.01 percent sodium hypochlorite did not improve the condition of roots held at 40° or 50°, it appears that decay was sufficiently controlled by these lower temperatures to preclude any effect by the disinfectant.

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ARIZONA AGRICULTURAL EXPERIMENT STATION, TUCSON

AN EVALUATION OF SEVERAL FUNGICIDES FOR CONTROL OF ROOT ROT OF CROFT LILIES[1]

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Summary

Terraclor, captan, and Copper Omadine have shown only partial control of certain soil-borne fungus diseases that cause rot and discoloration of lily bulbs, roots, and bulb scales. Isolations made before planting time revealed that many bulbs were infected with only one disease, while others carried pathogens of several diseases.

Fungicides were applied either as soil mix treatments or as direct dusting of the dampened bulbs. Terraclor and captan produced taller plants from bulb dusting than from soil mix treatments. Root systems received highest rating in the captan dust treatment plot. When combined with the basic fungicide, the miticide Thimet improved the rating in 8 of the 11 plots.

Disease-free bulbs flowered several days earlier than dusted bulbs, which flowered up to 3 weeks earlier than bulbs in soil mix treatments.

Absence of uniform distribution of infection by the several identified diseases made conclusions unwarranted with respect to most treatments for disease control. However, the soil mix treatments appeared to be superior to the dusted bulb treatments for control of the two most prevalent fungi, Cylindrocarpon radicicola and Fusarium solani.

External inspection did not reveal the type or the extent of infection found within lily bulbs sold commercially.

INTRODUCTION

The Croft Lily was originally found to be disease-free and superior to imported lilies with respect to production, quality of flowers, and adaptability to culture for Easter. In the 1930's it was heralded in the floral industry. Its commercial acceptance is now dependent upon the control of several soil-borne fungus diseases causing root injury, and bulb discoloration and rot during the growing and forcing periods.

In 1955, after working with lily bulbs having nematode-infection history, Jenson, Caveness and McWhorter (3) concluded that the danger of injury by parasitic meadow nematodes alone may be minimized as a major factor in the forcing quality of lily bulbs. Nematodes still play an important part, along with mites and symphlids, in the very complex problem of controlling the fungus diseases that attack roots, bulbs, and foliage of lilies (1, 4). Through injuries to root hairs and other plant tissues, nematodes assist in the invasion of plants by pathogens.

The complexity of the problem is increased because the climate of the large bulb-producing areas satisfies the disease preferences for temperature and soil moisture, although the diseases attack under widely varying circumstances. On one hand, high soil temperatures encourage Rhizoctonia solani and Fusarium oxysporum; while on the other, Pythium spp. and Phytophthora spp. prefer low soil temperatures (4, 5).

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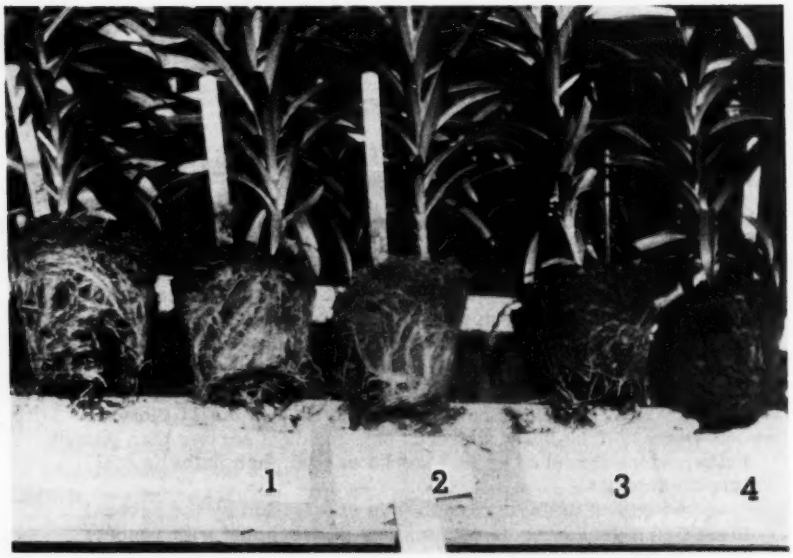

FIGURE 1. Rating or evaluation of Croft Lily root systems of the flowering plants. Highest rating #0, at left, shows no brown roots.

MATERIALS AND METHODS

In September 1957, the latest in a series of lily root rot studies was begun, when more than 600 Croft bulbs of known disease origin were received from the Pacific Northwest and placed in 40° F storage. Another shipment of 60 bulbs, sold commercially as disease-free, was also placed in storage. At 1-week intervals the bulbs were removed in lots of about 65 for inspection, tagged for identification, and then returned to storage, until the entire shipment had been indexed. From each numbered bulb several discolored scales were removed and given corresponding labels. The scales were then washed in distilled water, surface sterilized in Clorox, and the sample tissues transferred to potato-dextrose agar plates for culture. Each plate thus contained four or five isolations from one bulb. Fungi were identified after intervals of 5 to 10 days.

December 20, 1957, all bulbs except those showing no pathogens were potted in steamed soil mixtures and steamed 6-inch standard clay pots. Bulbs showing no pathogens were eliminated from further study. Bulbs that showed growth at potting time also were eliminated. The potted bulbs were staged inside other new 6-inch clay pots on gravel, on a raised bench in full sunlight, and grown at greenhouse night temperatures of 50° to 52° F for 3 weeks, then at 60°.

The potted diseased bulbs were arranged in 10 plots of 45 pots each, plus one plot of 57 disease-free bulbs.

The following pathogens were obtained from the bulb scales previous to potting: Colletotrichum lilii Plakidas, Cylindrocarpon radicicola Wr., Fusarium oxysporum Schlecht., Fusarium solani (Mart.) Appel & Wr., Penicillium spp., Rhizoctonia tuliparum Whet. & J. M. Arthur. In addition large and small mites were found.

February 20, 1958, root systems were inspected and evaluated for growth, coloration, and disease symptoms. April 9, 1958, the maturing root systems were again examined and evaluated for relative development and appearances with regard to disease symptoms. Samples of growing root tips were removed and cultured employing the same procedure as used before potting time, in the isolation and identification of disease pathogens. The following organisms were identified from the root tips: Cylindrocarpon radicicola Wr., Fusarium oxysporum Schlecht., Fusarium solani (Mart.) Appel & Wr., Sterile mycelium, Penicillium spp., Mites.

FIGURE 2. Sectioned white bulb showing well developed stem roots and bulblets. Internal brown rot is shown at the center of the bulb.

FIGURE 3. Sectioned bulb showing internal scale lesions and basal rot, and loss of basal and stem roots by fungus disease.

Table 1. Effect of chemical treatment of Croft Lily bulbs on disease control and certain growth characteristics.

Treatment[a]	Number of plants from which pathogenic organisms were isolated												Growth characteristics			
	Colletotrichum lilii		Cylindrocarpon radicicola		Fusarium oxysporum		Fusarium solani		Penicillium spp.		Rhizoctonia tuliparum		April root rating[c]	Average plant height (inches)	Average bud count (number)	Flowering date[d]
	S[b]	R[b]	S	R	S	R	S	R	S	R	S	R				
A	2	0	3	0	0	0	13	3	1	0	0	0	1.93	13.7	2.1	April 6
AT	5	0	3	0	0	0	6	2	0	0	1	0	2.06	15.4	2.6	late +
AP	3	0	10	4	1	1	8	1	2	0	2	0	2.53	13.0	2.2	late +
B	0	0	8	1	0	0	8	0	2	1	2	0	2.60	10.2	2.5	late +
BT	3	0	6	2	1	0	9	0	3	0	0	0	1.66	11.7	2.3	late +
BP	3	0	12	3	0	0	7	2	1	0	0	0	2.60	12.4	2.8	late +
C	0	0	8	4	0	0	7	1	3	0	1	1	2.06	10.1	2.2	April 22
CT	1	0	6	3	1	1	9	1	3	1	1	0	2.53	11.4	2.5	April 22
CP	4	0	4	3	0	1	8	0	4	0	0	0	2.80	10.3	2.8	April 18
D	0	0	8	4	0	1	9	2	2	0	1	0	2.80	8.5	2.0	late +
DT	2	0	6	2	0	2	6	1	2	2	2	0	1.40	10.9	2.2	late +
DP	3	0	4	5	1	0	8	0	0	1	3	0	1.86	11.2	2.8	late +
E	0	0	8	6	0	0	8	1	1	2	1	0	2.00	10.5	2.4	April 20
ET	3	0	3	2	1	1	9	1	2	0	2	0	1.80	12.0	2.7	April 22
EP	5	0	9	5	0	0	8	1	1	0	0	0	2.40	10.0	2.7	April 20
F	3	0	5	5	0	2	6	1	3	1	2	0	2.60	8.0	1.5	April 22
FT	7	0	4	3	1	1	4	0	0	0	3	0	1.53	10.9	2.5	April 20
FF	4	0	6	3	2	0	5	1	5	0	0	0	1.93	10.0	2.8	late +
G	0	0	4	5	0	0	11	3	3	0	0	0	2.06	11.4	2.6	late +
GT	1	0	7	2	0	0	3	2	0	1	2	0	2.13	12.0	2.5	late +
GP	6	0	3	3	2	0	5	1	1	0	3	0	2.26	13.4	3.0	April 6
H	4	0	8	3	0	2	7	2	1	1	0	0	2.06	15.1	3.7	March 28
HT	0	0	0	2	0	0	0	1	0	0	0	0	1.04	16.7	3.7	April 1
HI	0	0	0	1	0	0	0	2	0	0	0	0	1.94	15.0	3.9	March 28
K	2	0	4	2	0	3	8	1	0	2	2	0	1.86	18.5	2.7	April 6
KT	7	0	4	4	0	4	7	2	0	2	0	0	1.60	17.6	3.4	April 6
KP	3	0	6	5	1	2	6	2	4	2	1	0	1.06	16.8	2.5	April 6
L	0	0	5	5	0	3	5	3	0	3	1	0	2.40	16.4	2.9	April 6
LT	4	0	2	1	0	3	6	2	0	1	2	0	1.73	16.3	3.7	April 6
LP	3	0	9	3	3	1	4	2	3	0	0	0	1.13	16.0	2.7	April 6
Dis	1	0	5	3	3	5	6	1	2	2	0	0	2.60	14.5	3.4	late +
Dis T	1	0	5	2	0	1	6	1	0	0	1	0	2.60	15.0	2.6	late +
Dis P	1	0	6	0	2	4	4	2	2	2	3	0	2.46	15.2	2.8	April 6

[a]Fungicidal and miticidal treatments as follows:
A - 20% Terraclor at 200 lbs. per A (2 lbs. per 1 cu. yd.) (99.5 gms per 3 cu. ft. soil mix)
B - 20% captan at 200 lbs. per A (99.5 gms per 3 cu. ft. soil mix)
C - 20% Terraclor at 100 lbs. per A (49.77 gms per 3 cu. ft. soil mix)
D - 20% captan at 100 lbs. per A (49.77 gms per 3 cu. ft. soil mix)
E - 10% Terraclor and 10% Copper Omadine, each at 100 lbs. per A (99.5 gms per 3 cu. ft.)
F - Copper Omadine 10% at 2 lbs. per 1 cu. yd.
G - 20% Terraclor and 20% captan, each at 100 lbs. per A (each at 1 lb. per cu. yd.)
K - Wet diseased bulbs dusted with 20% captan
L - Wet diseased bulbs dusted with 20% Terraclor
Dis. - Diseased bulb check
H - Healthy bulb check
T - One third each plot above also treated with 4% Thimet at 1.67 gms/cu. ft.
P - One third each plot above also treated with Parathion dip (2 oz/5 gal. - 30 min.)

[b]S = isolated from scales, Dec. 1957.
R = isolated from root tips, April 1958.

[c]0 - Vigorous growth, all white roots, no disease symptoms.
1 - Heavy growth, few to many roots, yellow to brown discoloration.
2 - Good development, many roots brownish.
3 - Fair root development, many brown roots.
4 - Poor development, most roots brown or necrotic.

[d]+ = Flowered after April 22, 1958.

Evaluation of the root systems was expressed in five ratings (0-4), when inspected about 8 weeks after potting the bulbs. These ratings are shown in Figure 1, and summarized in Table 1.

RESULTS

As shown in Table 1, bulbs dusted with either Terraclor or captan made superior lineal growth, averaging 15.6 to 17.6 inches in height, while the same materials used as soil mix treatments produced plants 8.5 to 10.1 inches high as a plot average. Highest bud count was shown by captan-Thimet dusted bulbs, Terraclor-Parathion soil mix treatment, and with Terraclor dust alone.

The disease-free bulb plot HT showed the highest root rating 1.04. In the diseased bulb plots the bulbs dusted with captan plus Parathion, KP, showed highest root rating, 1.06, with the Terraclor-Parathion dust treatment, LP, 1.13. Dusted bulbs, treated with either Terraclor or captan were earlier flowering than were bulbs in the soil mix treatments. Earliest flowering was observed in the disease-free plots H, HT, Hp, on March 28, April 1, and March 28, 1958, respectively. The earliest flowering diseased bulb plots, dust treatments, flowered April 6, 1958. Plots flowering late, after April 22, included, in general, the Terraclor soil mix treatments.

No single treatment showed satisfactory control of all diseases. Leaf scorch injury occurred in treatments with captan, used alone or in combination with Terraclor, Thimet, or Parathion. This observation was in agreement with Hildebrandt, Beck, and Reinert (2). At potting time the distribution of the various identified pathogens was not uniform throughout the plots. Conclusions with respect to disease control with most of the treatments would, therefore, be unwarranted. However, the soil mix treatments appeared to be superior to the dusted bulb treatments for control of the two most prevalent pathogens, Cylindrocarpon radicicola and Fusarium solani. The development of bulblets was inversely proportionate to the disease injury to stem roots.

After the flowering period, certain bulbs showing good color, firmness and apparently without external disease symptoms were sectioned. This revealed internal injury from brown rot, as shown in Figure 2.

In Figure 3 brown basal rot and scale lesions were conspicuous as internal symptoms, and external injury is shown in the destruction of basal and stem roots.

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EFFECTIVENESS OF NON-DIRTING CULTIVATION AND SOIL-SURFACE APPLICATIONS OF PCNB IN CONTROLLING PEANUT STEM ROT IN VIRGINIA

Kenneth H. Garren

In 1958 Garren and Duke (4) reported that for 3 years, under Virginia conditions, the deep covering of organic matter on land prior to planting peanuts (Arachis hypogaea) followed by "non-dirting," a type of cultivation that avoided bringing soil in contact with the plants, effectively controlled the stem rot caused by Sclerotium rolfsii Sacc. Recently the writer discussed the role of the herbicide dinoseb in this non-dirting cultivation (2).

During the second year of the Garren and Duke study Cooper (1) reported that the new fungicide PCNB (pentachloronitrobenzene) (7) was one of three fungicides that showed promise in the control of stem rot in North Carolina. Garren and Duke (3) used PCNB as a drench of 12 pounds of active ingredient per acre with conventional ("dirting") cultivation in 1956 and reported a significant decrease in percentage of plants infected with S. rolfsii for the drenched plots and a non-significant increase in yield. In 1958 the North Carolina Extension Service made the application of PCNB in mixture with the landplaster at pegging time an official recommendation for the control of stem rot (8). Harrison in 1958 (5) reported results of 2 years' tests in Texas, indicating that PCNB applied as a spray around the crown of peanut plants with each of three cultivations may control stem rot. He noted in 1959 (6) that in a 1-year commercial-field test PCNB dust worked into the soil in a 12-inch band over the seed row appeared effective against stem rot; and in a smaller randomized test, either a spray or a dust applied to the crown of plants or the incorporation of dust into the row at planting gave some control. In this test one application of sprays and dust gave as good control as split applications.

In the 1957 and 1958 growing seasons, an experiment was conducted at Holland, Virginia with non-dirting cultivation and dirting or conventional cultivation as the main plots and dinoseb herbicide and no herbicide as subplots. The sub-subplots were PCNB drench (12 pounds of active ingredient per acre), PCNB with the landplaster (15 pounds of active ingredient per acre), and no PCNB. PCNB in landplaster was applied on top of the row at the regular time for landplaster application, which is the time of full bloom. In order to be sure that some stem rot was present in the plots to be treated, the PCNB drench was applied 10 weeks after planting, the time previously determined as being 2 weeks prior to the maximum increment of stem rot. All plots were thinned to a comparable stand following emergence. Infected plants were recorded at weekly intervals and yield of pods was recorded at harvest. Data were subjected to analysis of variance.

Since the effect of the dinoseb treatment on stem rot and yield was discussed recently (2), and since there were no statistically significant first-order interactions either year, the discussion here is confined to the effect of PCNB and the cultivation procedures on infection and yield.

In 1957 only PCNB in landplaster significantly reduced proportion of plants showing obvious symptoms of stem rot (Table 1). In 1958 both PCNB in landplaster and PCNB drench significantly reduced obvious symptoms of stem rot (Table 1). Non-dirting cultivation was highly effective in reducing stem rot both years (Table 1). Superimposing PCNB on non-dirting cultivation did not appreciably further reduce infection.

In 1957 PCNB drench gave a significantly higher yield of pods than did PCNB in landplaster (Table 2). In 1958 there was no statistically significant difference in yield among the three treatments (Table 2). Difference in yield between dirting and non-dirting cultivation was not statistically significant in 1957 (Table 2). In 1958 yield from non-dirting cultivation was highly significantly superior to that from dirting cultivation (Table 2). Use of PCNB in landplaster in conjunction with non-dirting cultivation during the 2-year period resulted in reduced yield. Use of PCNB as a drench in conjunction with non-dirting cultivation resulted in slightly increased yield. On the other hand, PCNB had a definite tendency to increase yield when used with dirting cultivation.

Thus, although PCNB as applied was effective in reducing infection and tended to increase yield when used with dirting cultivation, its use in conjunction with non-dirting cultivation was of questionable value. There is, therefore, no basis at present for considering soil surface applications (applications not worked into the soil) of PCNB as either a substitute for or a supplement to non-dirting cultivation in a control program for peanut stem rot in Virginia (9).

Table 1. Effect of application of PCNB on prevalence of Sclerotium rolfsii infection on Virginia Bunch 46-2 peanuts cultivated in different ways, Holland, Virginia, 1957 and 1958.

Year and treatment	Prevalence of S. rolfsii (in percent)		
	Dirting cultivation	Non-dirting cultivation	Average
1957:			
PCNB drench	15.5	6.0	10.8
PCNB in landplaster	10.7	4.0	7.4
None (check)	15.0	8.3	11.7
Average	13.7	6.1[a]	9.9
1958:			
PCNB drench	11.5	2.8	7.1[b]
PCNB in landplaster	14.0	4.1	9.0[b]
None (check)	28.6	7.1	17.8
Average	18.0	4.7[c]	11.4
Both years:			
PCNB drench	13.5	4.4	8.9
PCNB in landplaster	12.4	4.1	8.2
None (check)	21.8	7.7	14.8
Average	15.9	5.4	10.7

[a]Significantly smaller at 5% level than dirting cultivation.
[b]Significantly smaller at 5% level than no PCNB.
[c]Significantly smaller at 1% level than dirting cultivation.

Table 2. Effect of application of PCNB on pod yield of Virginia Bunch 46-2 peanuts when plants were cultivated in different ways, Holland, Virginia, 1957 and 1958.

Year and treatment	Pod yield (pounds per acre)		
	Dirting cultivation	Non-dirting cultivation	Average
1957:			
PCNB drench	2923	2627	2775[a]
PCNB in landplaster	2739	2432	2585
None (check)	2712	2560	2636
Average	2791	2540	2666
1958:			
PCNB drench	2859	3164	3012
PCNB in landplaster	2905	3044	2974
None (check)	2583	3149	2866
Average	2782	3119[b]	2951
Both years:			
PCNB drench	2891	2895	2893
PCNB in landplaster	2822	2738	2780
None (check)	2647	2854	2751
Average	2787	2829	2808

[a]Significantly higher at 5% level than PCNB in landplaster.
[b]Significantly higher at 1% level than dirting cultivation.

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FURTHER STUDIES WITH STREPTOMYCIN FOR WILDFIRE CONTROL IN BURLEY TOBACCO PLANT BEDS, NORTH CAROLINA, 1958

Luther Shaw, G. B. Lucas, and John H. Wilson, Jr.[1]

Summary

Excellent control of wildfire (Pseudomonas tabaci) on Burley tobacco in the plant bed resulted from four weekly spray applications of either streptomycin sulfate or streptomycin nitrate at concentrations of 200 ppm. Either two or three spray applications of streptomycin sulfate at a concentration of 400 ppm beginning 1 week after the plants were inoculated with P. tabaci effectively arrested the disease and prevented losses in stand. Post infection spray treatments with streptomycin sulfate appear to have considerable practical value in controlling wildfire in Burley tobacco plant beds. However, it is evident from these tests that the use of Tween 20 and growth-regulating substances in the mixtures containing streptomycin sulfate had little value in wildfire control.

INTRODUCTION

Wildfire control studies in Burley tobacco plant beds were continued at the Mountain Research Station, Waynesville, North Carolina, along the same general lines followed in 1955, 1956, and 1957 (2, 3, 4). A brief report of the methods employed and the results obtained in the 1958 studies are presented.

MATERIALS AND METHODS

Plant bed management practices, experimental design, inoculation of the plants with P. tabaci, spray schedules, method of application, and of disease index ratings were essentially the same as those employed in previous years (2, 3, 4). Materials used for the first time included Tween 20, indole-3-acetic acid, and naphthyl acetamide in combination with streptomycin sulfate (Table 1). The use of the plant-growth-regulating substances was suggested by the work of Hemphill and Goodman (1) who found increased effectiveness in the control of fire blight (Erwinia amylovora) on apples with antibiotics by the addition of small quantities of certain growth-regulating substances to the spray mixture.

RESULTS

Weather conditions were more favorable for wildfire development during the course of this experiment than they were during the previous years (1955-1957) that similar tests were conducted. The disease appeared in epidemic proportions in the check plots about 10 days following inoculation. Considerable reductions in stand were evident in the check plots.

The disease index readings (Table 1) show that all of the treatments tested, with the exception of the combination of Tween 20 and linseed oil, gave excellent wildfire control in comparison with the check plots. It is of special interest to note that two and three spray applications of streptomycin sulfate at a concentration of 400 ppm starting 1 week after inoculation of the plants with the wildfire pathogen resulted in a high degree of disease control. The disease did not cause any reduction in plant stands following these treatments. These results have considerable practical significance in that they offer Burley tobacco growers a method of arresting the disease and saving their plant beds after wildfire infection has taken place. There is, however, no evidence that these post infection treatments completely eradicate the disease from the beds.

Indole-3-acetic acid and naphthyl acetamide at concentrations of 20 ppm in combination with streptomycin sulfate at a concentration of 100 ppm definitely did not lower the bactericidal properties of streptomycin sulfate. Unfortunately, a check treatment using streptomycin sulfate alone at a concentration of 100 ppm was not included in this test. Hence, the full effects, if any, from these growth-regulating-substances in accelerated disease control cannot be determined from these data. However, it is evident from these tests that the use of Tween 20 in

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Table 1. Wildfire control with varied concentrations and schedules of applications of streptomycin sprays and other materials in Burley tobacco plant beds, North Carolina, 1958.

Treatment and concentration	Method of application	Rate per 100 sq. yds. (gallons)	Number of applications	Disease index June 10
1. Streptomycin sulfate 200 ppm	Spray	3-5	4	4.6**
2. Streptomycin sulfate 200 ppm + maneb 1/2 lb/100 gal.	"	3-5	4	4.0**
3. Streptomycin nitrate 200 ppm	"	3-5	4	5.6**
4. Streptomycin sulfate 400 ppm	"	3-5	1[a]	11.4**
5. Streptomycin sulfate 400 ppm	"	3-5	2[a]	4.8**
6. Streptomycin sulfate 400 ppm	"	3-5	3[a]	3.2**
7. Bordeaux mixture 3-4-50	Drench	25	2	4.8**
8. None (check)				26.4
9. Tween 20 (1-2000) + linseed oil(1-100)	Spray	3-5	4	17.6
10. Streptomycin sulfate 100 ppm + indole-3-acetic acid 20 ppm	"	3-5	4	6.4**
11. Streptomycin sulfate 100 ppm + indole-3-acetic acid 20 ppm + Tween 20 (1-2000)	"	3-5	4	7.6**
12. Streptomycin sulfate 100 ppm + naphthyl acetamide 20 ppm	"	3-5	4	8.1**
13. Streptomycin sulfate 100 ppm + naphthyl acetamide 20 ppm + Tween 20 (1-2000)	"	3-5	4	10.8**
14. None (check)				18.5
LSD .05				4.9
.01				6.5
CV (%)				36

[a]The first application was made 1 week after the beds were inoculated with P. tabaci.
**Significant at the 1 percent level over the lowest check.

the mixtures containing streptomycin sulfate and growth-regulating substances had little value in wildfire control.

Neither blue mold (Peronospora tabacina) nor anthracnose (Colletotrichum sp.) appeared in these beds.

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UNITED STATES DEPARTMENT OF AGRICULTURE, AGRICULTURAL RESEARCH SERVICE, AND NORTH CAROLINA STATE COLLEGE

THE USE OF CHEMICAL SOIL DRENCHES TO CONTROL PHYTOPHTHORA PARASITICA VAR. NICOTIANAE AND THEIR PHYTOTOXIC EFFECTS ON TOBACCO AND TOXICITY TO OTHER SOIL FLORA[1]

O. D. Morgan[2]

Summary

Effective control of Phytophthora parasitica var. nicotianae was obtained with seven chemicals applied as drenches. The chemicals: Omadine derivatives, disulfide, zinc, iron, and manganese controlled the fungus at 300 and 600 ppm; N521 (Mylone) at 300 and 400 ppm; chemical 5400 at 600 ppm; and Orthocide 406 at 300 ppm. Chloropicrin treated controls were used. Control of 95 to 100 percent was obtained with the above treatments. All inoculated untreated controls died in 2 weeks. Changes occurred in the soil pH during the tests. The chemically treated soil had the same or higher pH than the control. Plants in Orthocide and chloropicrin treated soil were taller but had smaller leaves than the healthy controls. All other chemicals stunted tobacco planted 4 days after treatment. Tobacco planted 3 weeks after treatment grew normally. Population counts were made in the laboratory of the saprophytic bacterial and fungal flora for treatments listed above. Orthocide and chloropicrin gave a significant increase in bacterial flora, while all other chemicals showed either a decrease, the same, or higher counts than the untreated control. All fungus population counts were significantly lower than the untreated controls. Differences occurred between treatments and within treatments. The counts indicate that the chemical treatments lower the fungus population and lessen competition for bacterial growth. Differences in bacterial counts between treatments indicated some stimulation of bacterial growth by the chemicals probably had taken place. Changes in the saprophytic soil flora had little effect on the control of P. parasitica var. nicotianae. Laboratory tests on this fungus also showed that it was killed when exposed for 7 days to various rates of some of the chemicals, but not at 4 days. N521 killed the fungus at or above 80 ppm and the Omadine salts, disulfide, zinc, and manganese at or above 50 ppm killed the fungus. The iron Omadine salt at 200 ppm, chemical 5400 and Orthocide at 400 ppm were fungistatic. All the chemicals killed and prevented germination of the zoospores at low concentrations. The zoospores appear to be the weakest link in the life cycle. Of the chemicals used, N521 showed best promise for control of P. parasitica var. nicotianae.

Black shank is an important disease in Maryland, affecting tobacco in the field and plant bed. Chemical soil treatment studies were initiated in 1954 (9) to find a fungicide that would control the fungus Phytophthora parasitica Dast. var. nicotianae (B. de Haan) Tucker, the cause of black shank. Several factors were explored: 1) control of black shank by chemical soil treatments as drenches, 2) phytotoxic effects of the chemicals on tobacco, 3) residual effects and time to plant after treatment, 4) laboratory tests on the effects of the chemicals on the mycelia, sporangia, and spores of the fungus, 5) effects of the treatments on other fungi and bacterial flora of the soil.

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A number of attempts have been made to control black shank by treating the soil with chemicals. Nabam and formaldehyde was used by Heggestad et al. (2) to eradicate black shank from the field around diseased plants, while Valleau et al. (13) controlled black shank in the field with nabam, formalin, and copper sulfate. Nusbaum and Chaplin (10) were able to reduce the incidence of black shank in the resistant variety Dixie Bright 101 with methyl bromide. Black shank can be controlled in the plant bed with methyl bromide and chloropicrin, according to Lucas and Moore (7). Todd and Lucas (12) were able to control black shank in the plant beds with urea and calcium cyanamide combination, calcium cyanamide alone, and methyl bromide. The chemicals methyl bromide, vapam, nabam, and formaldehyde were used by Lautz (6) as soil drenches to control black shank. Kendrick and Middleton (3) controlled several root rot organisms with N521.

Preliminary in vivo and in vitro tests with a large number of chemicals known to have some fungicidal properties showed that a few of them gave some promise as soil drenches to control black shank (9). This report is a study of seven of these chemicals.

MATERIALS AND METHODS

The chemicals studied were Omadine (2-pyridinethione 1-oxide) derivatives disulfide, zinc, iron, and manganese; compound N521 (mylone) (3,5-dimethyltetrahydro-1,3-5, 2H-thiadiazine-2-thione); experimental fungicide 5400 (alpha-alpha-trithiobis (N-dimethylthioformamide); Orthocide 406 wettable (N-trichloromethylmercapto-4-cyclohexene-1,2-dicarboximide); and chloropicrin. All the chemicals except N521 and chloropicrin were 50 percent dusts. N521 was a 90 percent dust and chloropicrin was a liquid. Chloropicrin was used as a control chemical in the greenhouse. It was not used in laboratory tests.

Greenhouse tests were run in 4-inch clay pots in temperature chambers at 80° to 85° F. The average weight of the soil in each pot was 577 grams. The soil at the time it was weighed was wet enough to support plant growth. The soil was heavily infested with Phytophthora parasitica var. nicotianae. Five randomized replicates of four pots each for each chemical and rate were used. All pots were placed in clay saucers to prevent contamination from bench soil or other pots. The various concentrations of each chemical were based on the active ingredient and weight of the soil. Each chemical was added to enough water to drench aliquot 50 ml quantities on each of the 20 pots. Chemicals were poured on the potted soil once during the tests. Water was drenched on 20 untreated uninoculated controls and 20 untreated inoculated controls. Preliminary greenhouse tests had given some indications as to the rates of each chemical to be used. The soil was watered once each day for the first 4 days after treatment to filter the chemicals through the soil. Six-weeks-old tobacco seedlings, variety Maryland Catterton, were transplanted to the treated pots. The soil was watered thereafter as often as needed. Chloropicrin, a standard soil treatment chemical known to control black shank (7), was used as a control treatment. The soil was fumigated with chloropicrin for 7 days then aerated for 7 days before planting. Preliminary tests had indicated that some chemicals, at high rates, injured tobacco if applied too close to planting time. Another series of pots with inoculated soil and treated with the chemicals 5400, Orthocide 406, and N521 at 600, 300, and 400 ppm, respectively, was run along with treatments made 4 days prior to planting. In this series of pots tobacco was planted 3 weeks after treatment, instead of 4 days, to determine whether a later planting gave better growth at the higher rates. Plant stand counts were made at 3, 5, 7, and 9 weeks after planting. Growth measurements of the plants, distance from soil to growing tip, were made 5, 7, and 9 weeks after planting. The pH of the soil was determined before treatment and again at the end of the tests.

To determine the time of planting after treatment with the Omadine salts, a test on phytotoxicity was set up. A series of 20 four-inch pots containing disease-free soil was treated with the highest rates of the chemicals that caused stunting of the plants when planted 4 days after treatment. The first four pots were planted 2 weeks after treatment with 6-weeks-old plants. Four pots were planted each week thereafter for 5 weeks. Growth measurements were made on each set of four plants after they had grown for 4 weeks.

GREENHOUSE RESULTS

Results of the various tests are given in Tables 1 and 2. Table 1 shows the number of plants living out of 20 at the end of the counting periods. Ninety-five to 100 percent control was obtained with the two highest rates of the Omadine salts, the highest rates of N521, and chloropicrin. Chemical 5400 and Orthocide 406 maintained control at the highest rate in the

plantings made 3 weeks after treatment but not in plantings at lower rates made 4 days after treatment. Chloropicrin apparently killed the fungus. Chemical 5400, Orthocide 406, and N521 had a longer residual control effect than did the Omadine salts at the lower concentrations, as shown by the rate at which plants died, Table 1. Plants were dying in the third week at the lower concentrations, indicating that the chemicals were fungistatic. Apparently the fungus became active after the chemicals were leached out of the soil. Scheffer and Haney (11) found that Orthocide 406 and several of the Omadine salts were not effective after several weeks in the soil. Results here show that after 9 weeks all chemicals at the highest concentrations were still effective. Orthocide 406, N521, and chemical 5400 gave some control at the lowest concentrations. Untreated inoculated controls died within 2 weeks.

Soil pH changes (Table 1) after treatment at the two highest rates in the plantings made 4 days after treatment indicate that the pH increased as the concentration of the chemicals increased except in soil with the zinc Omadine salt. The pH levels of the high rate of all of the chemicals except Orthocide 406 were higher than the controls. The pH changes in the soil that were in favor of the fungus had little effect on incidence of the disease at the higher rates. Kincaid and Gammon (4, 5) showed that the incidence of black shank increases with increase of pH. The pH in these tests was offset by the fungicidal effects of the chemicals.

Table 2 gives results of measurements of growth rate. All the Omadine salts at 300 and 600 ppm, chemicals 5400 at 400 ppm, and N521 at 300 ppm considerably retarded growth of tobacco after 9 weeks when planted 4 days after treatment. Graham (1) showed that N521 retarded growth of tobacco. Growth in Orthocide 406 and chloropicrin treatments was as good as or better than the controls. However, plants exposed to the latter two chemicals had smaller leaves than the control. Difference in height was due to an increase of internodal length. Examination of the roots in the Omadine salts showed that most of the roots had not penetrated more than 1/2 of the soil after 9 weeks, indicating that the chemicals had a long residual effect at higher concentrations or that the roots were permanently damaged after initial exposure to the chemicals. Root growth in soil treated 4 days before planting with chemical 5400, N521, and Orthocide 406 was considerably better than growth in the Omadine salts but slightly less than the controls. Orthocide 406 and chloropicrin appeared to have stimulated plant growth, as did the plantings made 3 weeks after treatment with chemicals 5400, N521, and Orthocide 406. Table 2 also shows differences in growth rate between treatments and rates in treatments at the highest concentrations of the chemicals after 5, 7, and 9 weeks. Growth rate differences at the lower concentrations are not reliable because of the death of many of the plants and infection of the roots.

The average growth of the tobacco planted 2 weeks after soil treatment with 600 ppm of each Omadine salt and measured 1 month later indicated that 2 weeks was not enough time in which to prevent injury. Growth of treated plants was less than that of untreated plants. Tobacco planted 3, 4, and 5 weeks after treatment produced about the same growth as the untreated controls. Growth in soil treated with the iron and manganese Omadine salts was slightly better than the controls after the third week. Results indicate that tobacco should be planted in soil treated with the high rates of the Omadine salts at least 3 weeks following treatment.

LABORATORY TESTS

Laboratory tests were conducted to determine the efficacy of the chemicals without the soil and to determine how closely greenhouse and laboratory tests were correlated. The in vitro tests were made with the same chemicals used in the greenhouse except where otherwise indicated. The Petri-plate dilution method of Mason and Powell (8) was used. Sterile oats were used as media for fungus culture. Four Petri plates were prepared containing one inoculated seed each. Twenty-ml quantities of the various concentrations of the chemicals were then poured into the plates. After 2 days at 85° F measurements were made of the longest mycelial growth from the seed. After 4 days observations were made on the production of sporangia, spores, and spore germination. Untreated controls in sterile water were used to obtain growth rates on oat seed media without treatment. After 4 days the oat seeds were removed, washed in sterile distilled water, and placed in Petri plates containing sterile distilled water. After 5 days in sterile water observations were made on mycelial growth, production of sporangia and spores, and spore germination. If at the end of 5 days in sterile water no growth occurred seeds were placed on young tobacco leaves in a moist chamber. Moist chambers were Petri plates containing wet filter paper. Any infection of the leaf indicated that the chemicals did not kill the fungus. A 4-day period was time enough for infection

Table 1. Effects of soil drenches at different rates on control of Phytophthora parasitica var. nicotianae on tobacco.

Treatment and rate	Stand counts at 2-week intervals starting at the 3rd week[a]				
	3 weeks	5 weeks	7 weeks	9 weeks	pH, 9th week[b]
Omadine salts					
Disulfide					
25 ppm	14	4	0	0	
75 ppm	20	12	10	0	
150 ppm	20	12	8	0	
300 ppm	20	20	20	20	5.1
600 ppm	20	20	20	20	5.5
Zinc					
25 ppm	14	12	0	0	
75 ppm	20	12	9	0	
150 ppm	20	7	4	0	
300 ppm	20	19	19	19	5.4
600 ppm	20	20	20	19	5.4
Iron					
25 ppm	20	10	0	0	
75 ppm	19	12	9	0	
150 ppm	20	11	3	0	
300 ppm	20	20	20	20	4.8
600 ppm	20	19	19	19	5.0
Manganese					
25 ppm	18	10	0	0	
75 ppm	20	8	7	0	
150 ppm	20	12	7	0	
300 ppm	20	20	20	20	5.0
600 ppm	20	20	20	20	5.3
N521					
75 ppm	20	16	14	11	
150 ppm	20	20	18	13	4.5
300 ppm	20	20	20	19	4.9
400 ppm[c]	20	20	20	20	
Chemical 5400					
100 ppm	18	14	12	7	
200 ppm	20	20	15	5	4.6
400 ppm	20	20	20	16	5.0
600 ppm[c]	20	20	20	20	
Orthocide 406					
50 ppm	18	14	12	8	
100 ppm	20	20	18	10	4.4
200 ppm	20	19	17	15	4.5
300 ppm[c]	20	20	20	20	
Chloropicrin					
2 oz.	20	20	20	20	4.4
Untreated inoculated control	0	0	0	0	4.8
Untreated uninoculated control	20	20	20	20	4.4

[a]The first count was made 1 week after planting and 10 days after treatments.
[b]The pH of the soil at the start of the test was 4.8.
[c]A separate series planted 3 weeks after treatments counts were made the same time that other stand counts were made.

Table 2. Effects of chemical soil drenches on tobacco in soil inoculated with Phytophthora parasitica var. nicotianae.[a]

Treatment	5 weeks		7 weeks		9 weeks	
	plants living	cm growth	plants living	cm growth	plants living	cm growth
Omadine salts						
Disulfide						
300 ppm	20	5.4	20	8.0	20	13.9
600 ppm	20	3.6	20	4.4	20	7.7
Zinc						
300 ppm	19	5.4	19	7.7	19	12.3
600 ppm	20	5.5	20	6.2	20	8.4
Iron						
300 ppm	20	8.7	20	12.8	20	16.1
600 ppm	19	7.1	19	10.6	19	13.2
Manganese						
300 ppm	20	6.5	20	11.1	20	18.3
600 ppm	20	5.9	20	8.6	20	13.9
N521						
150 ppm	20	11.2	18	21.39	13	29.9
300 ppm	20	7.7	20	18.8	19	24.2
400 ppm[b]	20	20.5	20	27.5	20	34.5
Chemical 5400						
200 ppm	20	11.3	15	19.6	5	19.4
400 ppm	20	7.3	20	10.4	16	11.7
600 ppm[b]	20	17.0	20	24.2	20	29.3
Orthocide 406						
100 ppm	20	17.7	18	26.2	10	31.2
200 ppm	19	14.5	17	24.2	15	31.2
300 ppm[b]	20	25.6	20	31.8	20	41.2
Chloropicrin						
2 oz.	20	18.6	20	28.9	20	38.0
Untreated Uninoculated Controls[c]	20	11.7	20	21.6	20	30.9

[a]Same plants were used as those in Table 1.
[b]Treatments were made 3 weeks before planting.
[c]Although the height of the plants was less in the controls the plants had considerably larger foliage. Treatments apparently stimulated elongation of the stalk.

Table 3. Control and effects on mycelia, sporangia, spore production, and spore germination of certain chemicals on P. parasitica var. nicotianae in culture.

Treatment	Before washing seed		After washing seed
	mm growth after 2 days	Spore production, germination 4 days[a]	Spore production, germination 5 days[a]
Omadine salts			
Disulfide			
5 ppm	2	SD	SAG
20 ppm	0	none	SAG
40 ppm	0	none	SD
60 ppm	0	none	SD
Zinc			
5 ppm	2.5	SD	SAG
20 ppm	1	none	SAG
40 ppm	0	none	SAG
60 ppm	0	none	SAG
Iron			
5 ppm	5.5	SA	SAG
20 ppm	3	SA	SAG
40 ppm	0	none	SAG
60 ppm	0	none	SAG
Manganese			
5 ppm	3	none	SAG
20 ppm	0	none	SAG
40 ppm	0	none	SC
60 ppm	0	none	SC
N521			
5 ppm	1.5	SD	SAG
20 ppm	0	SD	SAG
40 ppm	0	none	none[b]
100 ppm	0	none	none[b]
150 ppm	0	none	none[b]
Chemical 5400			
5 ppm	4	SA	SAG
10 ppm	1.5	SD	SAG
25 ppm	0	none	SAG
50 ppm	0	none	SAG
100 ppm	0	none	SAG
Orthocide 406			
10 ppm	3.5	SD	SAG
40 ppm	2	none	SAG
80 ppm	0	none	SAG
120 ppm	0	none	SAG
175	0	none	SAG
Control	7	SAG	SAG

[a]SD=spores rare, SC=spores occasional, SB=spores frequent, SA=spores abundant, G=germination, and O=no growth.
[b]Seed put on leaf of tobacco. Infection occurred at 40, 100, 150 ppm.

Table 4. Effects of chemical drenches on the population of the bacterial and fungus flora in nutrient media 5 weeks after soil treatment.

Treatment	1-20,000 dilution Bacterial colonies (average of 4 plates)	1-5,000 dilution Fungus colonies (average of 4 plates)
Omadine salts		
Disulfide		
300 ppm	80	6.0
600 ppm	49	9.5
Zinc		
300 ppm	72	5.5
600 ppm	103	5.5
Iron		
300 ppm	118	6.0
600 ppm	126	11.0
Manganese		
300 ppm	127	10.0
600 ppm	69	8.5
N521		
150 ppm	101	6.5
300 ppm	20	3.0
Chemical 5400		
200 ppm	314	0.0
400 ppm	271	1.0
Orthocide 406		
100 ppm	512	7.5
200 ppm	450	5.0
Chloropicrin 2 oz.	451	6.5
Untreated control	69	46.0
L.S.D. at 1%	466.7	9.5
L.S.D. at 5%	340.2	6.9

of the leaf to take place. Similar tests were made in the Petri plates with chemicals at higher concentrations for 7 days, after which they were washed with sterile water. If no growth occurred after 7 days in sterile water, seeds were placed on tobacco leaves as before.

Dilution plates were made on Czapeks agar medium for fungus and bacterial colony counts to determine effects of the chemicals on soil flora. The dilution for bacteria was 1-20,000 and for fungi 1-5,000. One-gram samples were taken from each of 10 pots of soil from the two highest rates of chemicals in tests where plantings were made 4 days after treatment. The 10-gram samples were taken 5 weeks after planting at 2-inch depths, then mixed, and a 1-gram sample of this mixture was added to 100-ml sterile distilled water blanks. Four plates were made for each dilution and chemical concentration. The 5 weeks period was chosen because at this time there was an indication that the chemicals were being leached out of the soil. Also, at 5 weeks a few plants were dead in the higher concentrations and it was assumed that the population of the soil flora was increasing.

LABORATORY RESULTS

Table 3 presents results of laboratory tests to determine the effects of chemicals on mycelial growth, sporangia and spore production, and spore germination. Growth of mycelia was less in all chemical concentrations than in the control after 2 days. After 2 days, the mycelia did not increase significantly in any chemical and no further measurements were made. The seven chemicals used in these tests apparently act on the fungus in the same way. These chemicals inhibit the mycelial growth, production of the sporangia and spores, and spore germination. As long as the chemicals were present in the 4- and 7-day treatments the chemicals inhibited spore germination at all concentrations. Results indicate that none of the chemicals killed the fungus after exposure for 4 days. After 5 days in sterile distilled water, growth occurred on seed exposed to all concentrations of the chemicals except N521 at 40 ppm and above. Spore germination occurred after 5 days in sterile water in all concentrations of the zinc and iron Omadine salts, chemical 5400, Orthocide 406, and the control. No germination occurred in the disulfide at 30 ppm or above and in manganese at 40 ppm or above. Infection occurred in leaves on which inoculated seeds from 40 to 150 ppm of N521 were placed, indicating that all of the chemicals were ineffective at the concentrations and exposure time used.

Where the fungus was exposed to all the chemicals for 7 days then rinsed and observed 7 days later, no growth occurred in the Omadine salts and N521 at 50, 100, 150, and 200 ppm and none occurred in chemical 5400 and Orthocide 406 at 100, 200, 300, and 400 ppm. Average growth of the untreated control was 9 mm in 7 days. Since no growth occurred on the seed in any chemical at the rates given all the seeds were placed on tobacco leaves. After 4 days on the leaf, infection took place at all concentrations in the iron Omadine salt, chemical 5400, and Orthocide 406. No leaf infection occurred at any concentration with the disulfide, zinc, manganese Omadine salts, and none in N521 above 50 ppm. There were indications that chemicals 5400 and Orthocide 406 would control the fungus above 400 ppm. At 400 ppm half the leaves were not infected. This concentration was comparable to that used to control Phytophthora parasitica var. nicotianae in the soil. Fungus growth from the untreated control seeds infected tobacco leaves in 4 days.

When spores were produced in low concentrations of the chemicals they would swim for a time and come to rest, but they would not germinate so long as chemicals were present; these spores probably were killed. After the seed and the inoculum was exposed to the chemicals for 4 days, then washed, new spores were produced which would germinate. No sporangia or spores were produced after fungus exposure to the chemicals for 7 days, then washed and put in sterile distilled water for 7 days.

From the results observed, not all the chemicals killed the fungus, even at concentrations above those necessary to inhibit growth when the fungus was exposed at periods shorter than 7 days. The three Omadine salts, disulfide, zinc, and manganese at or above 50 ppm, and the chemical N521 at 80 ppm or above, killed the fungus when exposed to the chemicals for 7 days. Chemicals at concentrations lower than 50 ppm were either fungistatic, did not penetrate the seed, or they were not in contact with all the fungus long enough to kill it. Seed penetration and contact with all the fungus apparently requires a period longer than 4 days.

Table 4 shows results of colony counts of bacteria and fungi from 1-gram samples from pots of soil used in the test given in Table 1. All chemicals significantly controlled the fungus flora. There was no significant control of the bacterial flora at 5 weeks and most of the treated soils had a greater number of bacteria than the control; thus indicating that, by eliminating

fungus competition, bacterial population increased. Two chemicals had a lower count; the Omadine disulfide salt at 600 ppm and N521 at 300 ppm. There was no significant difference between concentrations of the chemicals in either the bacterial or fungus counts. Fungi predominantly found were species of Trichoderma 85 percent, species of Aspergillus, Penicillium, and Rhizopus 12 percent; miscellaneous fungi such as Botrytis sp., Streptomyces sp., and others made up the remaining 3 percent. No attempt was made to identify the bacteria that were present.

DISCUSSION

The chemicals studied gave excellent control of Phytophthora parasitica var. nicotianae with one drenching at relatively high rates. To obtain a more complete picture of the chemical control of a soil-borne fungus, other factors such as pH, phytotoxic effects on plants, effects on the fungus, effects on other soil flora, method of treatment, time of treatment, and residual effects should be considered. In most instances the pH changed as the concentration of the materials changed -- usually in favor of a pH that allowed better growth of the fungus. However, the chemicals were strong enough fungicides to offset this factor. Since most of the chemicals were phytotoxic at concentrations that controlled the fungus, treatments should precede plantings. Orthocide 406 was the only treatment that was not phytotoxic when plantings were made as close as 4 days after treatment. The rest of the chemicals should be applied at least 3 weeks before planting.

Effects of the chemicals on the fungus under study should be determined. All the chemicals tested in the laboratory, without the soil factor, inhibited mycelial growth, sporangia, spore production and germination. Probably the greatest effect was to inhibit germination of spores at all concentrations used in the laboratory tests. This appears to be a weak link in the life cycle. As indicated in the results, the disulfide, zinc, and manganese Omadine salts and N521 were the only chemicals that penetrated the seed and killed the fungus. Kill was obtained with the Omadine salts at 50 ppm and above and with N521 at 80 ppm and above. These salts killed only after long exposure (7 days) of the fungus to the chemicals. Of the chemicals used in this study, N521 is probably best. It possesses a wide range of activity as a fungicide, herbicide, and nematocide. Generally higher rates are needed to gain control of the fungus in the soil than in laboratory tests. The chemicals may inhibit mycelia, sporangia, spores and spore germination in the soil as they did in the laboratory, although this has not been shown. Penetration of the seed in the laboratory by some of the chemicals was necessary to obtain complete control. Penetration of roots and debris was necessary to obtain complete control of the fungus in the soil. A better picture is obtained of what is happening in the soil by running laboratory tests in conjunction with greenhouse soil tests. There is an apparent control of the fungus in the oat seed when the fungus is inhibited; however, when the seeds are placed on tobacco leaves, infection sometimes occurs, indicating that the fungus was still alive. The tobacco leaf test is a more conclusive test of the control or lack of control by the chemicals. Effects of the chemicals on other soil flora show that they are definitely fungicidal but have little or no bactericidal activity.

Greenhouse and laboratory tests both indicated that if the controlling concentration is maintained, the chemicals inhibit the fungus; but, as soon as the chemical is leached out or removed, the fungus may become active again. Laboratory tests may indicate whether a chemical is fungicidal or fungistatic.

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A PATHOGENIC STRAIN OF ERYSIPHE GRAMINIS F. SP. TRITICI ATTACKING ASOSAN WHEAT[1]

H. R. Powers, Jr., J. F. Schafer, and R. M. Caldwell[2]

Summary

A newly observed strain of Erysiphe graminis f. sp. tritici was compared with previously reported races from this country and Europe, and tentatively designated as race 2A. Race 2A was distinctive primarily because of its ability to attack Asosan wheat. However, other varieties which have been utilized as sources of powdery mildew resistance, such as Hope, Michigan Amber Selection, and Suwon 92, were also susceptible. Inoculation of host varieties with known genotypes indicated that race 2A had a genotype for pathogenicity of $V_aA_cA_tA_u$.

An unusual strain of Erysiphe graminis DC. f. sp. tritici Em. Marchal was obtained in Indiana during the summer of 1958. It was found to be distinct from races described by Taylor et al. (5) or Lowther[3]. When only the reactions of the standard wheat differential varieties were considered, this new culture seemed identical to one described by Powers and Moseman (3). However, in contrast to the latter culture, the Indiana strain was highly pathogenic on the

Table 1. Seedling reactions[a] of differential varieties of wheat to races 2 and 2A of Erysiphe graminis f. sp. tritici.

Host		Race	
Variety	Genotype[b]	2[c]	2A
Carsten V	$ml_cml_tml_u$	4	4
Salzmünde Stamm		4	4
Red Fern, Ulka, C.I. 11478	$ml_cml_tMl_u$	0	0
Axminster, C.I. 8195, Norka, C.I. 4377	$ml_cMl_tml_u$	0	0
Normandie, C.I. 12747	$ml_cMl_tMl_u$	0	0
Halle 13471		0	
Weihenstephan M_1, C.I. 13398		0-1	0
Chul, C.I. 2227	$Ml_cml_tml_u$		0
Axminster x Chul #83	$Ml_cMl_tml_u$		0
Asosan, C.I. 12665			4
Hope, C.I. 8178			3
Michigan Amber Sel., C.I. 11770			4
Suwon 92, C.I. 12666			3

[a] 0 = immune, 1 = highly resistant, 2 = moderately resistant, 3 = moderately susceptible, 4 = very susceptible.
[b] Data from Carter (1), Powers and Sando (4) and unpublished data from Powers and Sando.
[c] Data from Nover (2).

[1] Joint contribution of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the Department of Botany and Plant Pathology, Purdue University Agricultural Experiment Station. Journal Paper No. 1432, Purdue University Agricultural Experiment Station.
[2] Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and Professors of Plant Pathology, Purdue University, respectively.
[3] Unpublished data of the late C. V. Lowther.

variety Asosan, C.I. 12665[4]. Asosan has not been used as a differential variety since it was consistently resistant to all cultures of E. graminis f. sp. tritici to which it had been exposed. This variety was included in the Uniform Wheat Mildew Nursery from 1949 through 1955 and was found to be among the most resistant entries each year. Since the Indiana culture attacks Asosan, it must have a genotype distinct from that of any race to which Asosan has previously been exposed.

The Indiana culture was used to inoculate eight of the nine varieties[5] of wheat that Nover (2) used to distinguish races of E. graminis f. sp. tritici occurring in Germany. As seen in Table 1, this culture gave results almost identical to those of Nover's race 2. Data on the reaction to race 2 of Asosan and other varieties included in the present test are not available, since these varieties were not included in Nover's tests. Therefore, the Indiana culture was tentatively designated as race 2A, indicating virulence on Asosan. Race 2A was also found to be virulent on Hope, C.I. 8178; on Michigan Amber Selection, C.I. 11770; and on Suwon 92, C.I. 12666. These varieties, together with Asosan, have been used by several plant breeders as sources of powdery mildew resistance. Race 2A is therefore potentially important and deserves more than usual attention.

The host genotypes for mildew resistance presented in Table 1 are based primarily on data by Carter (1) involving the study of five genes for mildew resistance. Three of these genes, Ml_c, Ml_t, and Ml_u, condition resistance to mildew in all stages of plant growth. Investigations by Powers and Sando (4) supported Carter's findings and also extended this work to include studies of the genotypes of the pathogen. Data from genetic studies[6] and also from inoculations with cultures with known genotypes have indicated that Asosan possesses a single, dominant gene conditioning mildew resistance which is distinct from those described by Carter.

It has been found in the case of powdery mildew of wheat (4) that for each gene pair conditioning response in the host there is a complementary gene in the parasite conditioning pathogenicity. Therefore, on the basis of the reactions of Asosan, Axminster, Chul, and Ulka to race 2A, it was possible to formulate a genotype for it. If the symbols A and V are used to indicate avirulence and virulence and subscripts a[7], c, t, and u to identify the complementary host genes, the genotype for race 2A can be designated as $V_aA_cA_tA_u$.

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[4]C. I. refers to accession number of the Cereal Crops Research Branch.
[5]Seed furnished by I. Nover, Martin-Luther-Universität, Halle-Wittenberg, Germany.
[6]Unpublished data from T. M. Starling, Associate Agronomist, Virginia Agricultural Experiment Station, Blacksburg, Virginia.
[7]Subscript "a" is proposed to indicate the gene for pathogenicity complementary to the gene found in Asosan.

INFLUENCE OF MOISTURE CONTENT AND TEMPERATURE UPON MOLD INVASION AND GERMINATION OF STORED WHEAT[1]

Thomas D. Wyllie and Clyde M. Christensen[2]

Summary

Samples of hard and soft red winter wheats from commercial storage and seed grade hard red spring wheat were stored at moisture contents of 15, 16, and 17 percent and temperatures of 20, 30, and 40° C, and tested for germination and percent of surface disinfected kernels yielding storage fungi after 2, 6, and 10 weeks. Using germination percentage, and percentage of surface disinfected kernels yielding storage molds as criteria of deterioration, the hard and soft red winter wheats from commercial bulks deteriorated much more rapidly than did the seed grade hard red spring wheat. In general, deterioration increased with increasing moisture content, increasing temperature, and increasing time.

INTRODUCTION

The work here reported was undertaken to determine whether different lots of wheat, when stored under the same conditions favorable to the growth of storage fungi, would deteriorate at different rates.

MATERIALS AND METHODS

Wheats: The soft red winter wheat (SRW) was obtained from a commercial bin in Maumee, Ohio and the hard red winter wheat (HRW) from a commercial bin in Omaha, Nebraska; both were No. 2 grade, had been in storage for approximately 6 months, contained few or no germ damaged kernels, and probably represented better than average samples of SRW and HRW wheats in commercial channels. The hard red spring wheat (HRS) was certified seed grown in Montana; it was almost free of storage fungi, without detectable damage, and in excellent condition.

Moisture Content: Moisture contents were determined by either the one-stage or two-stage air-oven method specified by Cereal Laboratory Methods (1) and are given on a wet weight basis.

Percentage of Kernels Infected with Storage Fungi: One hundred kernels were shaken 1 minute in 1 percent sodium hypochlorite, rinsed in sterile water, cultured on malt agar containing 7.5 percent sodium chloride, the dishes incubated 5 to 7 days, and the colonies counted and identified.

Germination: One hundred kernels were placed on moist blotting paper, kept at room temperature, and the germinated seeds counted after 5 to 7 days.

Preparation and Storage of Samples: Samples of grain were adjusted to the desired moisture contents by adding distilled water, stored at 5° C for 48 hours and shaken occasionally to permit uniform distribution of moisture. Portions of 200 gm each were then put in 16 oz. bottles closed with screw caps equipped with waxed paper liners. The volume of grain in each bottle amounted to less than half the volume of the bottle, not including the approximately 50 percent void space among the kernels. In the grain stored 6 to 10 weeks at 17 percent moisture it is possible that sufficient carbon dioxide accumulated to inhibit the growth of the molds and to reduce seed germination, but it is doubtful if accumulation of carbon dioxide had a major effect on the results obtained with the grain stored at lower moisture contents or for shorter periods of time. Samples at moisture contents of 15, 16, and 17 percent were stored at 20°, 30°, and 40°C and tested after 2, 6, and 10 weeks for germination and percentage of seeds invaded by fungi.

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[2] Research Fellow and Professor, respectively, Department of Plant Pathology and Botany, University of Minnesota.

RESULTS

Data on the original condition of the two samples of wheat are given in Table 1, and the results after 2, 6, and 10 weeks in Table 2 and in Figure 1.

Table 1. Percent germination, and percent of surface disinfected kernels yielding storage fungi, of two samples of wheat before storage tests were begun.

Wheat	Germination (percent)	Surface disinfected seeds yielding fungi (percent)
HRW	95	15
SRW	93	15
HRS	97	0-5

Table 2. Percent germination, and percent of surface disinfected kernels yielding storage fungi, of three samples of wheat stored 2, 6, and 10 weeks at moisture contents of 15, 16, and 17 percent and temperatures of 20°, 30°, and 40°C.

Temperature (°C)	Wheat	Moisture content (percent wet weight)	Weeks stored: 2		6		10	
			Percent germination	Percent seeds infected	Percent germination	Percent seeds infected	Percent germination	Percent seeds infected
20	SRW	15	89	10	72	9	75	20
		16	90	3	55	22	54	100
		17	89	58	21	91	4	100
	HRW	15	89	16	96	28	90	20
		16	92	14	92	57	52	100
		17	90	32	42	100	14	100
	HRS	15	99	11	99	2	97	0
		16	99	2	98	0	96	2
		17	96	6	95	22	89	62
30	SRW	15	93	16	75	25	30	48
		16	88	58	8	100	0	100
		17	63	97	0	100	0	100
	HRW	15	88	19	40	79	32	100
		16	82	100	16	100	0	100
		17	65	98	6	100	0	100
	HRS	15	95	6	97	4	94	2
		16	96	9	96	78	90	70
		17	98	72	50	100	15	100
40	SRW	15	27	30	8	100	0	100
		16	14	100	0	100	0	100
		17	7	100	0	100	0	100
	HRW	15	22	12	0	75	0	100
		16	15	0	0	95	0	100
		17	11	4	0	62	0	100
	HRS	15	96	10	94	10	50	5
		16	93	0	60	20	6	53
		17	88	97	0	100	0	100

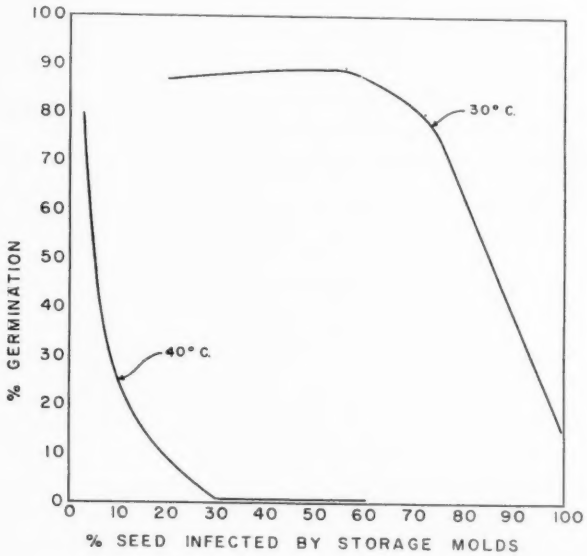

FIGURE 1. Relation of infection of kernels by storage molds to decrease in germination in grain stored at 30° C and 40° C.

FIGURE 2. Longitudinal section through the embryo end of a kernel of wheat from the sample stored 10 weeks at 17 percent moisture content and 40° C. No mycelium or sporophores are present on the outside of the pericarp, but the embryo is greatly shrunken and the space between the embryo and the inner side of the pericarp is filled with mycelium.

DISCUSSION

There were large differences in germination percentages and in percentage of surface disinfected kernels yielding fungi, between the SRW and HRW wheats from commercial storage on the one hand, and the seed grade HRS on the other. At 20°C and 16 percent moisture, for example, after 10 weeks the SRW and HRW wheats germinated 52 to 54 percent, and 100 percent of the surface disinfected kernels yielded storage fungi, while the HRS germinated 96 percent and only 2 percent of the surface disinfected kernels yielded storage fungi. The differences were even greater at 40°, 15 percent moisture, and 6 weeks. Apparently different lots of wheat stored under essentially the same conditions may deteriorate at greatly different rates. It is not implied that the differences are characteristic of the different classes of wheat or that they are inherent; recent tests in our laboratory have revealed considerable differences in rapidity of storage mold invasion of different samples of the same variety of HRS wheat. The differences here reported may be due in part to the degree to which the different lots of wheat had been invaded by storage molds before they were stored in our laboratory tests.

In general, at any one moisture content germination decreased, and percentage of surface disinfected kernels yielding fungi increased, with increasing time and with increasing temperature. In the HRW wheat at 40° C for 2 weeks the percentage of kernels yielding storage fungi was low, and the germination also was low, but this is about the only exception to the trend. Also, at any one temperature germination percentage decreased and percentage of surface disinfected kernels yielding storage fungi increased with increasing moisture content, and with increasing time. Evidently the storage fungi -- mainly members of the Aspergillus glaucus and A. candidus group species -- were able to develop fairly rapidly even at a temperature of 40°. Where grain is harvested in regions of high temperature, it is not unusual for it to be stored in bulk for some time at a temperature of 35° to 40° and at a moisture content of 15 percent. Judging by the results presented here, storage under those conditions for 6 weeks or more might result in extensive invasion by storage molds and great decrease in germination. From the mycological standpoint such grain would be considered to be partly deteriorated, although this deterioration would not be detectable by the ordinary inspection procedures used to determine the grade -- and thus the quality and price -- of grain. Freshly harvested wheat generally is free of any but light and superficial infections by storage fungi (3, 4), and even when the grain arrives at terminals it usually has not been invaded by storage molds to any great degree (2). Subsequent invasion of the stored grain by storage fungi may lead to extensive damage before any trouble becomes evident to the practical men, but tests that would measure the number and kinds of storage fungi present and the increase of these fungi during the storage period might help in evaluating storability of and damage to the grain. Such tests evidently now are being used by at least one firm as a regular part of their storage operations (2).

The dark germs or embryos typical of germ damaged or sick wheat were observed in a number of the samples, especially those stored 10 weeks at 40°C and moisture contents of 16 and 17 percent. In nearly all of these, no fungus was visible on the outside of the kernel, but the embryo was overgrown and the cavities within the embryo filled with mycelium. A typical example is shown in Figure 2.

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THE INHERITANCE OF NEW SOURCES OF OAT STEM RUST RESISTANCE[1]

J. Artie Browning and K. J. Frey[2]

Summary

Eight strains from the World Collection of Oats, C.I. Nos. 2413, 2710, 3030, 3031, 3037, 3038, 3039 and 3040, are resistant to race 8A of the oat stem rust fungus as well as to races 6, 7, 7A, 8 and 13A. The reactions of F_2 seedlings from crosses between Burnett and C.I. 2710, C.I. 3030 and C.I. 3031, to races 6, 7, 7A and 8 and of F_1 plants from crosses between Markton and the eight C.I. strains, to races 6, 7 and 8 suggest that the gene or genes responsible for the inheritance of resistance in these C.I. strains is or are recessive. C.I. 2710, C.I. 3030, and C.I. 3031 each possessed a single locus conditioning resistance to each of the stem rust races. The data were not adequate to distinguish whether the same locus conditioned resistance to all four races. The gene or genes from the C.I. strains do not condition resistance when plants are maintained at high (85° F) temperatures.

To date only one type of resistance to race 6 of the oat stem rust fungus (Puccinia graminis Pers. f. sp. avenae Eriks. & E. Henn.) has been used in the development of new oat varieties. This factor for resistance occurs in oat varieties Rodney and Canuck and was designated by the letters "BC" (10) (hereinafter referred to in this paper as the B gene). From a screening of the World Oat Collection Browning (3) found eight lines (C.I. Nos. 2413, 2710, 3030, 3031, 3037, 3038, 3039 and 3040) with resistance to race 6, which according to origin or parentage would most likely not possess the B gene.

The importance of new sources of resistance to stem rust takes on additional meaning with the recent discovery of races 6A (5), 8A and 13A (4, 8). The B gene does not condition resistance to these races, or to race 7A. Varieties possessing the Richland or A gene are resistant to race 7A while varieties containing the White Tartar or D gene are resistant to race 8A. There have been no known genes for resistance to races 6A and 13A.

The eight oat lines selected for race 6 resistance were found to be resistant to race 13A also (3). They are also resistant to race 8A (Table 1). Thus, these eight lines offer good to

Table 1. Reactions of seven varieties or strains of oats to five races of stem rust.

Variety or strain	Genes	Reaction to races				
		6	7	7A	8	8A
Richland	A	4	1	1	4	4
Rodney	B	1	1	4	1	4
White Tartar	D	4	4	4	2	2^+
Burnett	BD	1	1	4	1	2^+
C.I. 2710		1-X^-	2-X^-	X^--3^-	1^+-2^-	1^+-2
C.I. 3030		1-2	2-2^+	2^+-3^-	1^+-2^-	1^+-2
C.I. 3031		1-X^-	2^+-X^-	X^--3	1^+-2^-	1^+-2^+

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[2] Associate Professor of Plant Pathology and Professor of Agronomy (Farm Crops), respectively.

moderate resistance to races 6, 7, 7A, 8, 8A and 13A.

For the most efficient use of the sources of resistance possessed by these C.I. strains it is necessary to know their modes of inheritance. Consequently, crosses were made between three of the race 6-resistant lines found in the World Oat Collection and Burnett variety to ascertain whether some of the resistance genes were identical or allelomorphic to the B or D genes and, secondarily, to incorporate possible new resistance genes into varieties adapted to the Corn Belt. The race 6-resistant lines used were C.I. 2710, C.I. 3030 and C.I. 3031 (Table 1).

MATERIALS AND METHODS

Burnett variety of oats (C.I. 6537) carries two dominant genes for stem rust resistance, the B and D genes; thus it is resistant to races 6, 7, and 8 and susceptible to race 7A. The lines C.I. 2710, C.I. 3030 and C.I. 3031 are resistant to races 6, 7, and 8 but differ from Burnett in their reactions to these races and in being moderately resistant to race 7A (Table 1).

In the F_2 generation seedlings from crosses between Burnett and each of these three C.I. strains were tested for reaction to stem rust races 6, 7, 7A and 8. The seedlings were inoculated with a talc suspension of spores of the individual races, wet down with a 1/2 percent solution of Tween-20, kept in a moist chamber overnight, and subsequently kept in a 70° to 75° F greenhouse. The plants were classified for reaction to rust 12 days after inoculation according to the system of Bailey (2). Infection types of 1, 2, or X^- were pooled as resistant and X, 3 and 4 were pooled as susceptible reactions.

RESULTS

The observed and expected numbers of seedlings in the resistant and susceptible classes calculated according to certain genetic hypotheses are presented in Table 2 for each cross and

Table 2. Expected and observed numbers of resistant and susceptible F_2 oat seedlings.

Cross	Race	Reaction	Observed	Expected	Ratio	X^2	Probability
Burnett X C.I. 2710	6	R	33	28.4	13:3	3.92	.02-.05
		S	2	6.6			
	7	R	24	26.0	13:3	0.82	.30-.50
		S	8	6.0			
	7A	R	3	5.8	1:3	1.75	.10-.20
		S	20	17.2			
	8	1^--1	16	18.0	3:1	0.89	.30-.50
		1^+-2	8	6.0			
Burnett X C.I. 3030	6	R	111	108.1	13:3	0.43	.50-.70
		S	22	25.9			
	7	R	162	--	--	--	--
		S	188				
	7A	R	11	16.3	1:3	2.27	.10-.20
		S	54	48.7			
	8	1^--1	19	22.5	3:1	2.17	.10-.20
		1^+-2	11	7.5			
Burnett X C.I. 3031	6	R	133	134.1	13:3	0.05	.80-.90
		S	32	30.9			
	7	R	87	92.6	13:3	1.83	.10-.20
		S	27	21.4			
	7A	R	14	17.8	1:3	1.06	.30-.50
		S	57	53.2			
	8	1^--1	51	50.2	3:1	0.05	.80-.90
		1^+-2	16	16.8			

rust race. The segregation for reaction to race 6 gives a satisfactory fit to a ratio of 13 resistant to 3 susceptible in all three crosses. A similar fit holds for reaction to race 7 except in the cross Burnett X C.I. 3030. Two independent pairs of genes appeared to be segregating in the five cases which fit a 13:3 ratio. In each of these cases Burnett contributed the dominant allele of the B locus that conditions resistance to races 6 and 7. The other parent in each of these cases contributed the recessive allele of a gene pair in which resistance was recessive. Only one gene was contributed from a C.I. strain in any given cross.

As few as one and as many as five loci could be involved in the segregation for reaction to races 6 and 7 in these three crosses. The present data do not provide a method for determining whether the same locus may be involved in these five cases. Such identification could best be made by studying the reactions of segregates from crosses between the C.I. strains. The similarity of reaction and segregation tend to indicate, however, that the same locus is involved in all of the C.I. strains and the same locus conditions reaction to races 6 and 7.

The reactions to race 7A of the segregates from the three oat crosses fit satisfactorily to a ratio of 1 resistant to 3 susceptible. This indicates that the resistance to race 7A found in either C.I. 2710, C.I. 3030, or C.I. 3031 is due to a single locus in which resistance is recessive. Again resistance to race 7A may be conditioned by the same locus in each of the C.I. strains or by a different locus in each one.

Two points are of considerable interest and importance relative to the segregation for reaction to races 6, 7 and 7A in the three oat crosses. First, the three C.I. strains do possess at least one gene, and possibly more, which was hitherto unknown. When present in a cross which also carried the B gene, segregation for reaction to races 6 and 7 occurred which shows that the new gene or genes was at a locus different from the B locus. The new gene or genes could be allelomorphic to the A gene from Richland but must be a different gene since resistance to race 7A was recessive in contrast to resistance being dominant with the A gene.

The second point is that this is the first case in which resistance to oat stem rust has been reported conditioned by a recessive gene. Similar instances are known in crown rust of oats (6, 7) and in stem rust of wheat (1).

To test further the hypothesis that resistance was due to a recessive gene from the C.I. strains, a number of F_1 plants resulting from crosses between Markton (a variety completely susceptible to oat stem rust) and the eight race 6-resistant C.I. lines from the World Oat Collection were inoculated with races 6, 7 and 8. All of the F_1 plants were susceptible. This shows that the resistance gene or genes was recessive and corroborates the above hypothesis.

Since two genes conditioning resistance to race 8 entered the crosses from Burnett, and possibly another gene or genes from the C.I parent, analysis of the F_2 data (Table 2) for resistance to this race was more difficult. The B gene from Burnett produces a type 1^--1 reaction to race 8 which is epistatic to the type 2 reaction conditioned by the D gene. Only two types of resistant reactions were distinguished in this test, type 1^--1 typical of Burnett and type 1^+-2^- from the C.I. parent. No F_2 plant was found susceptible to race 8.

Two possible conclusions could be drawn from these results: First, if the gene or genes for resistance from the C.I. parents conditioned resistance as a dominant and they were independent of the B or D genes from Burnett, the ratio would be as a minimum 63:1. With the small number of F_2 seedlings tested it would be possible to have no susceptible seedlings appear. If the new gene or genes reacted with resistance being recessive and inherited independently, as was the case with races 6 and 7, the expected ratio would be 48 type 1^--1: 13 types 1+ and 2: 3 susceptible. On this basis some susceptible seedlings should have been found. Yet none was found (Table 2), and in one test not reported in Table 2 (because data were not recorded in different resistant infection types) F_2 seedlings from the cross Burnett X C.I. 3030 gave a ratio of 120 resistant to none susceptible when tested with race 8. The second possible conclusion is that the gene or genes for resistance to race 8 from the C.I. strains was allelomorphic to either the B or D genes. In each of the three crosses the ratio of resistant types to race 8 was a satisfactory fit to 3 types 1^--1: 1 type 1^+-2^-. No accurate distinction could have been made between types 1^+ -$2^-$and 2 if the latter had appeared so the data do not indicate whether the gene or genes from the C.I. strains were allelomorphic to the B or D genes. If the same gene from a C.I. strain conditions resistance to races 6, 7, 7A and 8 the gene contributed could not be allelomorphic to the B gene since susceptible plants occurred when races 6 and 7 were used to study the segregation of the B and the unknown genes.

One cannot say from these data that the same unknown gene is responsible for reaction to all four races in all the new sources of resistance, but the susceptible reaction of F_1 plants between Markton and the C.I. lines to races 6, 7, and 8 is suggestive. The fact that the gene(s) conditioning resistance in these eight C.I. strains is, like the B gene (9), temperature sensitive

in that resistance is conditioned at moderate temperatures (75° F) but not at high temperatures (85°) (Browning, unpublished data) further suggests that these lines possess a single, common gene for oat stem rust resistance. Close linkage is another possibility.

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BLACK MESDAG, A POSSIBLE PARENT FOR OAT SPECIES CROSSING

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Summary

An apparent cross using Black Mesdag, a hexaploid oat, and a derived tetraploid, Abd. 101 (C. I. 7232), as parents had high fertility in the F_1, F_2, and F_3 generations. Also the seedling resistance to crown rust races 263, 264, 274, and 294 carried by the tetraploid was evident in the progeny of the cross. The apparent ready crossability of Black Mesdag should be helpful in making both interspecies and intraspecies crosses.

The crossing of diploid (7n) and tetraploid (14n) oats with the hexaploid (21n), or cultivated species, in order to transfer desired genes for disease resistance has posed serious difficulties. Crosses between oats differing in chromosome numbers usually produce very few viable seeds. Any possible aid in increasing the number of successful gene transfers would be helpful.

In making more than a thousand oat crosses during the past third of a century, the writer has observed that the degree of success differs with the varieties chosen as parents. The century-old Black Mesdag variety of hexaploid oats has seemed to be especially easy to hybridize.

The characters and the interesting history of Black Mesdag were reported by de Haan (2). With regard to the leading varieties grown in the Netherlands in the second half of the last century, he stated "Zwarte President (Black President or Mesdag oat), an early-maturing black oat, is the only variety of this group that has remained in cultivation, because of its low lime requirements and its resistance to copper deficiency. The history of Zwarte President can be traced back to 1860. At that time it was put on the market by J. DeJonge, the president of a drainage-district association [hence Black President]. It was grown especially in the region of 'peat colonies' and on newly reclaimed fen moors or heath. In France, De Vilmorin brought out a selection called Avoine Noire de Mesdag." Stanton (6) stated "Black Mesdag (C. I. 1877) was introduced into the United States from Vilmorin-Andrieux et Cie, Paris, France. It was selected by Louis de Vilmorin from Black President."

In the United States Black Mesdag has long been of special interest. Reed et al. (4) reported it to be highly resistant to all races of oat smut present in this country in 1925. H. K. Hayes of the Minnesota Agricultural Experiment Station crossed Black Mesdag with a Minota x White Russian selection in what is presumably the first attempt to produce stem rust-, smut-resistant oat strains in the United States. Minnesota Double Cross A resulted. This was used by Hayes and coworkers at Minnesota as one of the parents of Mindo, which in turn they used in breeding the Minhafer variety.

Black Mesdag, classed as A. sativa by Etheridge (1) and Stanton (6), is an early-maturing, midtall oat with midsized, fairly stiff culms. The equilateral panicle branches are long, drooping, and arise at 5-7 nodes. Spikelets generally have two florets. Grains are rather slender, dull black, the lower bearing a geniculate awn, dark-colored, and twisted in the lower part. The rachilla segment supporting the second floret bears numerous hairs.

The writer has crossed Black Mesdag with other varieties of Avena sativa and with A. byzantina, fatuoid forms from different A. byzantina varieties, and a steriloid-fatuoid oat type. In such crosses black kernel color of Black Mesdag was dominant to gray, red, yellow, and white and gave F_2 ratios of 3:1, 12:3:1, 48:12:1, and 48:15:1, depending on the color of the other parent. Crossed with two black oats, no segregation for lemma color resulted. The crosses were effected without difficulty, and complete fertility apparently was obtained in all.

Black Mesdag was crossed with the crown rust-resistant derived tetraploid Abd. 101, C.I. 7232: (Avena abyssinica C. D. 4549) x (A. strigosa C. D. 3820), at Aberdeen, Idaho in 1957,

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[1] Agronomist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture. The writer is indebted to M. B. Moore and F. K. S. Koo of the University of Minnesota, Institute of Agriculture, for their data on the reaction of these oats to races of crown and stem rusts, and to Harold G. Marshall, Agronomist, Crops Research Division, Agricultural Research Service, for his data on the reaction of certain of these oats to crown rust race 264. The 16 backcrosses mentioned were made by Clemmer Marcus, formerly of the Crops Research Division.

when the plants had reached a rather advanced stage of development. Five florets on each of five panicles were emasculated and pollinated. One seed was obtained from each of four panicles.

The four F_1 seeds were planted in the Beltsville greenhouse in the fall of 1957, and the resulting plants were backcrossed with Black Mesdag, and 16 seeds were produced. The four F_1 plants were reasonably vigorous and showed little evidence of sterility. This high fertility had not been expected, since the F_1 plants in a previous interspecies cross with Black Mesdag showed a high percentage of sterility. All of these F_1 plants had black kernels and twisted awns on all lower florets, as did the two parents (Figs. 1 and 2).

FIGURE 1. Plant of Black Mesdag (left); and the F_1 of cross X57BL Black Mesdag x Abd. 101: Avena abyssinica C. D. 4549 x strigosa C. D. 3820, grown in the greenhouse at Beltsville, Md., in 1957-58 (right).

The backcrosses were space-planted at Aberdeen, Idaho in 1958. They showed a wide range in maturity, in plant height (43-57 inches), in panicle length (10-15 1/2 inches), in number of culms per plant (12-28), and in straw diameter. One supposed backcross had noticeably orange-colored straw and another was very late, although the original parents are both early. All backcrosses appeared to be almost fully fertile, and generous seed supplies were harvested.

The seed from the F_1 plants was sown at Aberdeen, Idaho in 1958. All F_2 plants appeared highly fertile. One cross, X57BL, segregated into some plants bearing black and some gray kernels, whereas all plants of the other three populations bore black kernels. One panicle was removed from each plant and the remaining heads of each cross were harvested in bulk.

In the fall of 1958, the seeds from F_2 plants were offered to several experiment stations, and one cross (X57BL) was grown in the greenhouse at Beltsville. Data on crown rust resistance were obtained at St. Paul, Minnesota, where F_3 populations of three of the four crosses were grown. All the progenies of two crosses were susceptible to all races of both crown and stem rust used as inoculum. The third cross, X57BL, was susceptible to the stem rust races used, but the plants segregated for resistance to crown rust.

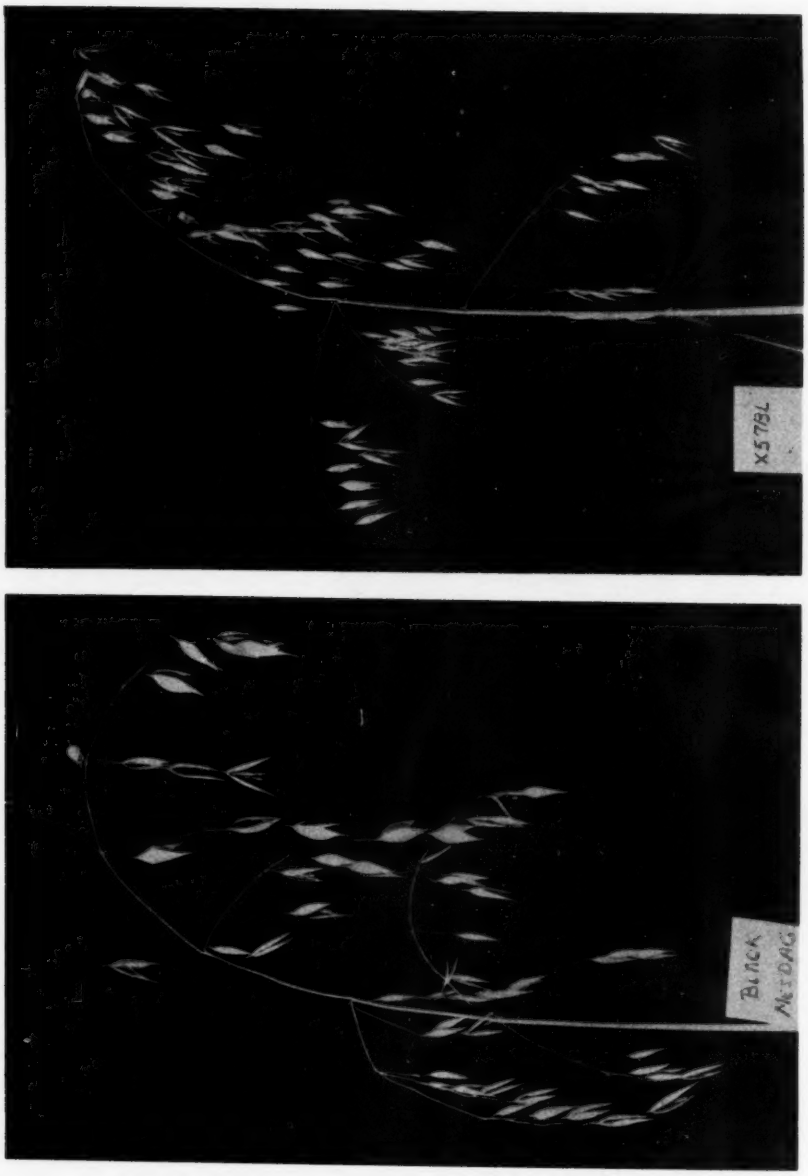

FIGURE 2. Panicles of Black Mesdag (maternal parent) (left) and the F1 plant of cross X57BL Black Mesdag x Abd. 101: Avena abyssinica x Avena strigosa (right).

Progenies were grown from 30 F_2 panicles from cross X57BL. Inoculations made in the seedling stage with spores of crown rust races 263, 274, and 294 revealed that segregation occurred in 27 of the 30 progeny populations; whereas one appeared presumably homozygously resistant and two completely susceptible to all three races. Four lines appeared resistant to races 263 and 294 and two additional lines to race 294. Eight lines appeared heterozygous for resistance to all three races, whereas about half of all lines appeared susceptible to race 274. The line that is apparently homozygously resistant to all three races is of special interest, as it would appear to be a fertile hexaploid oat.

The reaction of the rust tester varieties to the same crown rust races used to inoculate these hybrids at St. Paul are shown in Table 1.

Table 1. Reaction of oat varieties to three races of crown rust.

Variety	Reaction[a] to race 263	274	294
Anthony	S	R	R
Victoria	R	S	R
Appler	S	S	S
Bond	S	S	S
Landhafer	S	R	S
Santa Fe	S	R[b]	S
Ukraine	R	MS	MS
Trispernia[c]	MS	MR	MR
Bondvic	MR[d]	R	R
Saia	R	R	MR[d]

[a]S = susceptible; MS = moderately susceptible; R = resistant; MR = moderately resistant.

[b]One plant susceptible.

[c]In earlier readings appeared resistant to all three races but later readings indicated this was an error.

[d]Read R in earlier reading.

Seed of each of the four supposed backcrosses obtained, in which the F_1 of the Cross X57BL, Black Mesdag x Abd. 101, was used as the female and Black Mesdag as the male parent, was supplied to Harold G. Marshall of State College, Pennsylvania, in the spring of 1959. A report just received from him indicates that segregation as to reaction to crown rust race 264 occurred among the F_2 progeny of each of these four backcrosses but that the major percentage of the seedlings of each was found to be resistant to race 264. Black Mesdag proved fully susceptible and Abd. 101 completely resistant to race 264; hence, from these added observations, it would seem evident that lines of apparently fully fertile hexaploid oats that have resistance in the seedling stage to both the Landhafer- and Victoria-attacking races of crown rust have been produced.

DISCUSSION

Smut-resistant Black Mesdag has never shown resistance to either crown or stem rust so far as we know. The other parent of the cross X57BL, Abd. 101 (C. I. 7232) is apparently highly resistant to many races of both rusts. Murphy et al. (3) state with regard to C. D. 3820: "The Saia variety of A. strigosa has been used more widely in interspecific hybridization than C. D. 3820. The two varieties appear to be identical. They have been observed to have the same reaction to a number of races of both rusts and to other diseases and they appear to be identical for all agronomic characteristics." They stated further, "The parent variety of C. I. 7232, C. D. 3820, apparently was not homozygous for the genes conditioning resistance to races 6, 7, 7A and 8 of stem rust."

Zillinsky et al. (7) have given the history of the derived tetraploid Abd. 101 and report its reaction to different races of crown and stem rusts. Simons et al. (5) crossed the derived tetraploid, Abd. 101, with a susceptible tetraploid and found its resistance to race 264 of crown

rust was controlled by a single, almost completely dominant gene. It is especially interesting that Abd. 101 has resistance to stem rust as well as to crown rust, yet in the crosses reported in the present paper no resistance to stem rust has been observed so far in any of the segregates. That fact is admittedly something of a puzzle, but in the absence of any cytological information on these crosses of Black Mesdag x Abd. 101 no explanation for this apparent lack of stem rust resistance is attempted. So far no progenies of the cross X57BM have been studied for either crown or stem rust resistance; but data from St. Paul indicate that the other two crosses, X57BO and X57BP, having the same varietal parents as crosses X57BL and X57BM were susceptible to all races of both crown and stem rust used as inoculum.

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UNUSUAL OCCURRENCE OF SMALL-GRAIN DISEASES IN TEXAS IN 1957 AND 1958[1]

M. C. Futrell, I. M. Atkins, and C. D. Hobbs[2]

Summary

Small-grain diseases were heavy in Texas during the 1957 and 1958 growing seasons. During these years stripe rust became epiphytotic for the first time in history. Development of stripe rust in Texas shows similarity to the development of stripe rust on the Chengtu Plain of China. Septoria leaf blotch and scald of barley and Septoria leaf blotch, powdery mildew, and Helminthosporium culm rot of oats were observed in the State for the first time. Barley leaf rust and the two Septoria diseases of wheat were heavy in most of the small-grain-growing areas of the State. Stem rusts of wheat and oats were light during both years.

INTRODUCTION

Losses from small-grain diseases were major factors in production of small-grain crops in Texas in 1957 and 1958. These seasons were characterized by subnormal temperatures and average to slightly above normal rainfall over much of the State (Tables 1 and 2). The last severe outbreak of small-grain diseases was in 1949 (1), and the interim period was one of severe drought, when disease losses in small-grain crops were relatively low. Disease losses for each crop are shown in Table 3. A detailed report of losses (2) by agricultural extension districts of the State has been published as a mimeographed report.

EXPERIMENTAL OBSERVATIONS

Rusts

Stripe rust of wheat, Puccinia striiformis West., developed in the late winter at the Beeville and College Station areas where the mean maximum temperatures for February and March ranged from 6° to 10° F below normal in 1958 (Table 1). The disease spread northward through central and north-central Texas in March and April of both seasons but became epiphytotic in this area only in 1958. The mean maximum temperatures for the Waco-Denton-Wichita Falls areas ranged from 3° to 6° F below normal (Table 1) during March and April of both years. The disease continued to spread into the Texas Panhandle, where an epidemic occurred in both 1957 and 1958 during late April and May.

Leaf rust of wheat, P. recondita Rob., was present in the fall of 1956 and again in 1957 over most of the State. Very little winterkilling of the fungus occurred, so the disease increased slowly during the winter and spread rapidly during the cool moist spring. Losses were most severe in the north-central and Rolling Plains areas of the State. The Concho and Westar varieties were severely damaged, and races which can attack the Lee and Bowie varieties were present.

The most unusual development in oats was the sudden explosive buildup of the 213-216 race complex of crown rust, P. coronata var. avenae (Cda.) Fraser & Led., on varieties of Victoria parentage. Previously these races had been rare and large acreages of Victoria-derived varieties were protected from this disease. During both seasons crown rust became established in the Panhandle and spread southward across the State during the fall. The fungus became established and overwintered in the Gulf Coast Area and in northern Mexico. The disease spread northward across the State in the spring of 1957, causing severe losses in grain

[1]Texas Agricultural Experiment Station Technical Article No. 3185. The research work on which this report is based was conducted cooperatively by the Texas Agricultural Experiment Station and the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, College Station, Texas.

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Table 1. Seven-year mean maximum temperatures (°F) compared with 1957 and 1958 mean maximum temperatures at six locations in Texas.

LOCATION	February			March			April			May		
	Av.	1957	1958	Av.	1957	1958	Av.	1957	1958	Av.	1957	1958
Beeville	71	74	62	77	75	68	81	78	76	87	84	87
College Station	65	69	58	71	69	66	78	76	79	86	85	88
Waco	64	66	55	70	66	62	77	73	74	85	82	86
Denton	62	62	55	70	61	60	76	70	73	84	80	85
Wichita Falls	60	58	50	65	60	54	75	69	71	83	78	83
Amarillo	55	58	48	60	56	45	69	64	62	78	71	80

Table 2. Ten-year average rainfall (inches) compared with 1957 and 1958 mean rainfalls at six locations in Texas.

LOCATION	February			March			April			May		
	Av.	1957	1958	Av.	1957	1958	Av.	1957	1958	Av.	1957	1958
Beeville	1.7	1.2	6.3	1.1	2.2	.8	2.9	8.3	.6	3.0	9.1	1.8
College Station	3.2	2.7	2.7	2.6	5.7	1.6	5.0	12.5	4.2	4.1	3.5	2.0
Waco	2.4	2.4	3.3	2.2	5.6	1.0	4.0	13.4	3.6	5.2	7.6	5.6
Denton	1.9	3.4	0.3	2.1	5.7	3.8	4.5	12.2	6.2	6.0	16.8	3.5
Wichita Falls	0.9	1.6	0.6	1.4	2.4	2.5	3.1	8.5	2.5	6.3	12.0	4.4
Amarillo	0.6	1.1	0.6	0.8	2.8	2.4	1.3	2.7	1.7	3.7	4.4	2.5

Table 3. Losses from small-grain diseases in Texas in 1957 and 1958.

Disease	WHEAT				OATS				BARLEY			
	1957		1958		1957		1958		1957		1958	
	% Loss	Loss in bushels	% Loss	Loss in bushels	% Loss	Loss in bushels	% Loss	Loss in bushels	% Loss	Loss in bushels	% Loss	Loss in bushels
Leaf rust	3.9	1,468,111	2.7	2,292,067	11.2	5,821,000	5.2	2,553,841	1.12	61,068	1.2	108,636
Stripe rust	1.0	382,575	3.6	2,991,672								
Stem rust	0.7	245,157	tr	4,770	2.26	1,166,542	0.5	233,581	0.18	10,042	tr	
Septoria sp.	5.0	1,866,981	1.3	1,049,386	0.06	308,284	1.7	808,659				
Mildew	0.1	33,947		tr	0.17	89,898						
Loose smut	0.003	9,875	0.08	69,020	0.26[a]	134,524	0.05[a]	22,062				
Covered smut	0.001	3,220		tr								
Helminthosporium	tr		tr		13.65	7,059,696	3.1	1,520,150	5.18	281,850	1.0	85,185
Total all diseases	10.58	4,009,904	7.6	6,410,945	28.19	14,580,541	10.55	5,138,293	6.48	352,957	2.2	193,821
Total State Production		33,885,000		77,441,000		37,148,000		43,552,000		5,090,000		8,694,000

[a] Includes both smut species.

and forage. The disease became established again in 1958 and caused losses in forage in south Texas, but it failed to spread northward as in 1957. Heavy losses were restricted to the two southern tiers of counties adjacent to the Gulf Coast. Winter grazing seasons were cut short by 4 to 6 weeks in this area.

Barley leaf rust, P. hordeii Otth., was widespread in the State and caused the heaviest loss on record. Very few varieties showed resistance to the races present.

Stem rust, P. graminis Pers., of both oats and wheat was light during the 1957 and 1958 seasons. Infections on both crops could be found in trace amounts during the growing seasons, but damage was negligible. Studies on reaction of varieties and race identifications made by the Cooperative Rust Laboratory, St. Paul, Minnesota, showed that races 6, 7 and 7A were present on oats and that races 11, 15B, 17, 29, 38, 48A and 56 were present on wheat. The 17, 29, 48A complex was widely distributed on wheat in south and central Texas but did not move into the northern part of the State. The winter wheat varieties Comanche, Westar and Concho are resistant to these races. The temperatures that remained below normal in the central and northern part of the State (Table 2) may have limited spread of stem rust in the 1957 and 1958 seasons.

Helminthosporium Diseases

The Helminthosporium diseases of oats were destructive throughout most of the State in 1957 and were present but less destructive in 1958. The greatest damage was done by H. victoriae Meehan & Murphy on varieties of Victoria parentage. Studies of isolates from Southland, Bronco, and New Nortex varieties showed the presence of an H. sativum-like organism that produces a culm and crown rot on Southland and Bronco varieties and somewhat less severe symptoms on New Nortex. Helminthosporium leaf blotch, H. avenae Eidam, was observed in a number of fields.

Septoria Diseases

The two Septoria diseases incited by Septoria tritici Rob. and S. nodorum Berk. developed and caused damage on wheat in the north-central and Rolling Plains areas, in the very wet, cold spring of 1957, and were present in both areas in 1958 but caused somewhat less damage. During March, April, and May of 1957 rainfall was excessive (Table 2) in both of these areas of the State and temperatures (Table 1) were below normal. Both the Septoria diseases thrive under these conditions. The severe lodging that resulted increased the cost of harvesting and reduced the quality of the grain.

Septoria leaf blotch developed on oats in central and north Texas during both seasons. Strains of the pathogen which attacked a wide range of varieties were present at Denton.

Other Diseases

Net blotch, H. teres (Died.) Drechsl., became widespread and destructive on Goliad barley in south Texas. Scald was observed in central and north Texas, and some damage was caused by this disease in local areas. Powdery mildew, Erysiphe graminis avenae Em. Marchal, was observed on oats and caused some damage to pasture fields in south Texas during both seasons. Some damage was caused by powdery mildew, E. graminis tritici, on wheat in 1957 in north-central Texas, but the disease caused only minor damage in 1958. Barley powdery mildew was light during both seasons. The incidence of loose smut, Ustilago tritici (Pers.) Rostr., continued to increase with the increased acreage of the susceptible varieties Concho and Quanah. Other smuts of cereals were present and caused some damage.

DISCUSSION

The two stripe rust epiphytotics in Texas show similarities to the occurrence of stripe rust on the Chengtu Plain of China described by Ling (3). The wheat-growing areas in China and the United States fall between 28° and 36° N. latitude. The elevation of the wheat-growing area in China ranges from 1300 to 4000 feet, which is similar to that of the Rolling Plains and Panhandle of Texas. The 11-year mean temperature from February through May for the Chengtu Plain of China is compared with the 10-year average of the same months from 1949 to 1958 for the Wichita Falls, Texas, area (Table 4). Ling (3) pointed out that the heavy stripe rust epidemic of 1939 on the Chengtu Plain was due to 1) a large number of rainy days, 2) a

low amount of sunlight, 3) a low mean temperature, and 4) high relative humidity during April. These conditions produced very succulent wheat plants. The mean temperature at Wichita Falls was 4° F below normal in April 1957 and 1958. The number of cloudy days was high. The same conditions existed in the Texas Panhandle in late April and May, thus promoting the advance of the epiphytotic northward across the Wheat Belt of the United States.

Table 4. Mean temperatures (° F) for 1933-1943 on the Chengtu Plain of China and for 1949-1958 at Wichita Falls, Texas.

LOCATION	Feb.	March	April	May
Chengtu	47	55	63	72
Wichita Falls	46	53	63	72

A number of factors could be responsible for the occurrence of the stripe rust epidemic in Texas. Among these are 1) increased acreage and shifting of varieties in northern Mexico, and 2) buildup of inoculum in south and central Texas where temperatures were below normal during the winter months. These data would indicate that establishment of stripe rust is not limited by temperature alone on the Rolling Plains and Texas Panhandle; moisture, light, relative humidity, and amount of inoculum also may be limiting factors. Southward in central and south Texas high temperatures may be limiting factors, thus reducing inoculum being blown into the northern part of the State.

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RELATIVE SUSCEPTIBILITY OF SEVEN VARIETIES OF RED CLOVER TO DISEASES COMMON IN WISCONSIN

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Abstract

The relative susceptibilities of Chesapeake, Dollard, Kenland, Lakeland, Midland, Pennscott, and Wisconsin Common red clovers to powdery mildew, northern anthracnose, Stemphylium leaf spot, Cercospora leaf spot, rust, blackstem, the virus complex, and a few other diseases were assessed at Madison, Wisconsin from 1953-1958. Lakeland proved most resistant to powdery mildew and Lakeland and Dollard were most resistant to northern anthracnose. All varieties were susceptible to each of the other diseases, but Pennscott averaged least susceptible to Stemphylium and Cercospora leaf spots and Lakeland to blackstem and the virus complex. Kenland was most susceptible to rust, Dollard and Lakeland to Stemphylium leaf spot, and Pennscott to northern anthracnose.

INTRODUCTION

With the recent release of several new red clover varieties, interest in their relative reactions to common diseases has increased. This report summarizes some of the pertinent data recorded at Madison, Wisconsin from 1953-1958, inclusive. Since all tests were made in the field under natural conditions without artificial inoculation, disease severity varied greatly from year to year and from one part of a growing season to another. None of the diseases was severe every year or at all times of any year. This fact is reflected in the relatively low average reactions obtained even for susceptible varieties. The data do show, however, that the varieties compared differed markedly in their reactions to several diseases.

SOURCE AND KIND OF DATA

All data except those reported in Table 3 are averages of three sets of notes per year for each of three to five nurseries. The first set was obtained in June, a few days before the first hay crop was removed; the second set during the last days of July or the first part of August, immediately prior to removal of the second hay crop; and the third about the middle of October. Most of the observations were made on stands in their first harvest year, but the October data from one new seeding each year were included in the averages. All the nurseries were sown broadcast in April or early May with oats as a companion crop. Each set of notes was based on three to six replicates in each nursery.

RESULTS

Powdery mildew (Erysiphe polygoni DC.): Lakeland was clearly superior to all other varieties in resistance to powdery mildew (Table 1). Never was more than an occasional plant of this variety infected with mildew, and during 1953-1958 the percentage of such plants in the population was reduced by selection. All other varieties were susceptible, but Dollard was somewhat less susceptible than five of the seven varieties compared.

Northern anthracnose (Kabatiella caulivora (Kirchn.) Karak.): Lakeland and Dollard were most resistant (Table 2). Pennscott was most susceptible, but Kenland was only slightly less susceptible. Midland and Wisconsin Common were intermediate. While it was not uncommon to obtain ratings of 5 or 6 in the more susceptible varieties in some seasons, Lakeland and Dollard never showed more than a trace of anthracnose in any of the Wisconsin tests.

The complete susceptibility of many red clover varieties to northern anthracnose is shown by some data recorded at Presque Isle, Maine[2] in 1956 (Table 3). The disease is only

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[2]This nursery was established by C. R. Blackmon, to whom the writer is indebted for permission to take the notes.

Table 1. Relative susceptibility to powdery mildew of 7 varieties of red clover at Madison, Wisconsin, 1953-58.

Variety	Disease reaction for indicated year[a]						
	1953	1954	1955	1956	1957	1958	Average
Lakeland[b]	1.0	0.2	0.2	0.1	0.0	0.0	0.2
Dollard	5.6	2.1	4.9	3.8	2.2	3.1	3.6
Chesapeake[c]	5.6	2.2	5.4	4.5	3.5	3.9	4.2
Kenland	6.0	2.3	5.2	4.7	3.6	4.2	4.3
Midland	5.4	2.4	6.0	5.1	3.5	4.4	4.5
Pennscott	5.8	2.4	5.9	4.9	3.6	4.4	4.5
Wisconsin Common[d]	5.8	2.4	6.2	5.0	3.3	4.1	4.5

[a]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe).
[b]Prior to its naming and release in 1959, this variety was tested under the name Wisconsin Synthetic.
[c]Prior to its naming and release in 1957, this variety was tested under the name Stevens.
[d]This strain, known in Wisconsin as Olds Medium, is a blend of Wisconsin strains obtained from the Olds Seed Company, Madison, Wisconsin.

Table 2. Relative susceptibility to northern anthracnose of 7 varieties of red clover grown at Madison, Wisconsin, 1953-58.

Variety	Disease reaction for indicated year[a]						
	1953	1954	1955	1956	1957	1958	Average
Lakeland	1.0	0.5	0.2	0.0	0.0	0.0	0.3
Dollard	1.0	0.5	0.8	0.0	0.3	0.0	0.4
Midland	2.3	1.2	2.1	0.5	0.9	0.9	1.3
Wisconsin Common	2.5	1.4	2.0	0.5	0.7	1.2	1.4
Chesapeake	3.3	1.8	1.9	0.8	1.6	2.1	1.9
Kenland	4.0	2.8	3.0	1.2	2.4	2.4	2.6
Pennscott	4.0	3.1	3.2	1.7	2.6	2.4	2.8

[a]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe).

Table 3. Reactions to northern anthracnose of 11 red clover varieties grown in the Uniform Red Clover Disease Nursery at Presque Isle, Maine in 1956[a].

Variety	Disease reaction on July 26, 1956[b]
Lakeland	1.0
Dollard	2.0
LaSalle	4.7
Purdue	5.0
Midland	6.7
Chesapeake	7.0
Kenland	8.0
Pennscott	8.0
Sanford	8.0
Port Gibson	8.3
Reinholdt	8.3

[a]This nursery was seeded in 1955. It included three replicates and had not been cut when the data were recorded.
[b]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe -- plants completely brown).

Table 4. Relative susceptibility to Stemphylium leaf spot of 7 varieties of red clover at Madison, Wisconsin, 1953-58.

Variety	Disease reaction for indicated year[a]						
	1953	1954	1955	1956	1957	1958	Average
Pennscott	2.6	1.4	2.3	1.2	4.2	1.6	2.2
Chesapeake	2.6	1.4	2.2	1.3	4.6	1.8	2.3
Kenland	2.5	1.5	2.3	1.3	4.4	1.6	2.3
Wisconsin Common	2.6	1.5	2.2	1.3	5.1	1.6	2.4
Midland	2.8	1.6	2.5	1.9	5.2	1.6	2.6
Lakeland	2.4	1.5	2.0	2.2	6.3	2.1	2.8
Dollard	2.6	1.6	2.7	2.2	6.2	2.4	3.0

[a]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe).

Table 5. Relative susceptibility to Cercospora leaf spot of 7 varieties of red clover at Madison, Wisconsin, 1953-58.

Variety	Disease reaction for indicated year[a]						
	1953	1954	1955	1956	1957	1958	Average
Pennscott	3.3	3.2	2.2	4.9	1.7	1.5	2.8
Chesapeake	3.3	3.6	2.2	5.1	1.7	1.5	2.9
Dollard	3.3	3.7	2.3	5.2	1.6	1.3	2.9
Kenland	3.3	3.4	2.2	5.0	1.8	1.5	2.9
Lakeland	3.3	3.4	2.3	5.4	1.5	1.3	2.9
Wisconsin Common	3.4	3.5	2.2	5.3	1.8	1.5	3.0
Midland	3.7	3.6	2.1	5.6	1.7	1.5	3.1

[a]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe).

Table 6. Relative susceptibility to rust of 7 varieties of red clover at Madison, Wisconsin, 1953-58.

Variety	Disease reaction for indicated year[a]						
	1953	1954	1955	1956	1957	1958	Average
Dollard	0.8	0.2	1.0	0.4	1.4	2.6	1.1
Pennscott	0.8	0.2	1.2	0.4	1.1	2.7	1.1
Lakeland	0.8	0.2	1.2	0.4	1.4	2.8	1.1
Midland	0.8	0.2	1.4	0.4	1.3	3.4	1.2
Wisconsin Common	0.8	0.2	1.3	0.4	1.2	3.3	1.2
Chesapeake	0.8	0.4	1.4	0.4	1.4	3.4	1.3
Kenland	0.8	0.4	1.9	0.4	1.3	4.0	1.5

[a]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe).

infrequently as severe as this at Madison, Wisconsin.

Stemphylium leaf spot (Stemphylium sarcinaeforme (Cav.) Wiltshire), and sometimes S. botryosum Wallr.): All varieties were susceptible to Stemphylium leaf spot; but Dollard and Lakeland were more susceptible than the other varieties (Table 4). This disease is usually not very important in stands cut twice for hay. It is more important in new seedings, where it sometimes becomes abundant by late fall, and in fields left for seed.

Cercospora leaf spot (Cercospora zebrina Pass.): Midland and Wisconsin Common had slightly higher average disease ratings than the other five varieties, but all were susceptible (Table 5).

Rust (Uromyces trifolii (Hedw. f. ex DC.) Lév.): When red clover is cut twice during a growing season, rust usually does not become abundant until rather late in the fall, when moderate amounts may develop on the aftermath. None of the seven varieties was resistant to rust; each showed a reaction of 5 or more at some time during the tests. Kenland, on the average, was more susceptible than the other varieties (Table 6).

Ordinarily rust is most important in new seedings and in stands left for seed. When it is

Table 7. Relative susceptibility to blackstem of 7 varieties of red clover at Madison, Wisconsin, 1953-1958.

Variety	Disease reaction for indicated year[a]						
	1953	1954	1955	1956	1957	1958	Average
Lakeland	1.2	0.8	1.0	1.3	1.3	0.6	1.0
Dollard	1.2	1.2	1.0	1.3	1.3	0.6	1.1
Kenland	1.2	1.1	1.1	1.3	1.3	0.6	1.1
Pennscott	1.2	1.0	1.0	1.3	1.5	0.6	1.1
Wisconsin Common	1.2	1.1	1.1	1.3	1.4	0.6	1.1
Chesapeake	1.3	1.3	1.0	1.3	1.5	0.6	1.2
Midland	1.7	1.0	1.1	1.3	1.4	0.6	1.2

[a]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe).

Table 8. Relative susceptibility to the virus complex of 7 varieties of red clover at Madison, Wisconsin, 1953-58.

Variety	Disease reaction for indicated year[a]						
	1953	1954	1955	1956	1957	1958	Average
Lakeland	3.0	1.9	1.5	2.2	2.5	2.3	2.2
Dollard	4.2	2.3	1.6	2.3	2.4	2.0	2.5
Kenland	3.2	2.6	1.7	2.6	2.8	2.5	2.6
Chesapeake	4.0	2.8	1.6	2.4	2.9	2.4	2.7
Pennscott	4.0	3.0	2.1	2.4	3.0	2.6	2.8
Midland	4.0	2.8	2.2	2.6	3.3	3.1	3.0
Wisconsin Common	4.0	2.8	1.8	2.7	3.1	3.4	3.0

[a]Based on a scale of 0 to 9 (0 = no disease; 9 = very severe).

severe in hay fields it is usually in those where cutting has been delayed beyond the proper time. There are exceptions, however. On June 16, 1949 the writer saw a field of red clover near LeRoy, Illinois that was just approaching the cutting stage, and it was severely rusted; stems as well as leaves were covered with pustules.

Blackstem (Ascochyta imperfecta Pk., also designated as Phoma trifolii E. M. Johnson & Valleau on red clover, and Cercospora zebrina Pass.): In Wisconsin, blackstem is seldom important on red clover except in stands left for seed. It is widely distributed, however, and may be found in most fields in trace amounts. Table 7 gives the average reactions to blackstem of the varieties included in the test. The severity level in all years was so low that it was not possible to ascertain differences in susceptibility between the varieties. Midland had most blackstem in 1953, but not in the other years.

Virus diseases (Several viruses[3]): Virus diseases and root rots were the most important maladies of red clover in Wisconsin and probably in the entire north-central region during 1953-1958. Table 8 summarizes the virus data obtained in Wisconsin. All varieties were susceptible but Lakeland ranked among the strains having the lowest disease ratings and Midland and Wisconsin Common ranked among those having the highest.

Root and crown rots (Several species of soil fungi): Root and crown rots are present wherever clovers are grown. In Wisconsin, Fusarium spp. have been most commonly associated with diseased red clover roots[4]. Most of these species are weak pathogens, requiring weakening of the host before they cause much damage. Under field conditions plants are weakened by drought, insects, nematodes, winter injury, and poor management. They may also be predisposed by being grown on land deficient in fertility or low in pH. Occasionally stands are plowed after the removal of a single crop of hay because of the combined effects of the root-rotting organisms and the predisposing factors. Varieties differ in susceptibility to single isolates of Fusarium, but none has been resistant to the entire range of isolates normally found in field soils when the plants were weakened or injured. Lakeland and Dollard have persisted longer

[3]Hanson, E. W., and D. J. Hagedorn. 1952. Red clover, a reservoir of legume viruses in Wisconsin. (Abst.) Phytopathology 42: 467.

[4]Kilpatrick, R. A., E. W. Hanson, and J. G. Dickson. 1954. Root and crown rots of red clover in Wisconsin and the relative prevalence of associated fungi. Phytopathology 44: 252-259.

than other varieties under Wisconsin conditions, but whether this greater longevity was due to superior resistance to root rots was not established.

Other diseases: Pseudopeziza leaf spot (Pseudopeziza trifolii (Biv.-Bern.) Fckl.) was moderately abundant in the northern half of the State in some years, but occurred sparingly in the southern part. No consistent differences in susceptibility of varieties were observed. It should be emphasized, however, that the severity level in the nurseries at Madison was probably too low to give a reliable evaluation of resistance. Williams[5] reported varietal differences in susceptibility to Pseudopeziza leaf spot and found that early varieties were damaged more by this disease than were later maturing varieties.

Sooty blotch (Cymadothea trifolii (Fr.) Wolf), bacterial blight (Pseudomonas syringae v. Hall), and blackpatch (Rhizoctonia leguminicola Gough & Elliott) occurred in some years but only in trace amounts. It was not possible to measure differences between varieties in reactions to these diseases.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND WISCONSIN AGRICULTURAL EXPERIMENT STATION

5Williams, R. D. 1927. Red clover investigations, 1919-1926. Univ. Coll. Wales. Welsh Plant Breed. Sta. Bull. Ser. H 7: 1-136.

THE EFFECT OF NEMATODES ON THE DEVELOPMENT OF ROOT ROT AND YIELD OF CANNING PEAS[1]

W. A. Haglund and Thomas H. King[2]

Abstract

In 1957 Dowfume MC-2 and Dorlone were applied to plots in three fields, and in 1958 Dorlone and Telone were applied to plots in one field, in which canning peas subsequently were grown. Dowfume MC-2 controlled root rot but Dorlone and Telone did not, although yield of peas was greater in all of the treated plots than in the untreated controls. The population of parasitic nematodes was reduced approximately the same amount by all three fumigants. Results of tests in the greenhouse, using soil from the fields fumigated in 1957, agreed with those obtained in the field.

INTRODUCTION

Nematodes influence the development of root diseases of tobacco, cotton and many other crops. Seinhorst (4) reported that Hoplolaimus uniformus Thorne, 1949 influences early yellowing of peas, and Labruyere and Seinhorst (3) determined that Fusarium oxysporum Schlecht. f. pisi (Linford) Snyd. & Hans. race 3 would not cause the early yellowing of peas in the absence of the nematode H. uniformus. Taylor et al. (5) reported that several species of parasitic nematodes were associated with canning peas in Minnesota and studies were undertaken in 1957 to determine if nematodes were part of the root rot complex of canning peas.

MATERIALS AND METHODS

In 1957, three fields were fumigated with Dowfume MC-2, a general biocide, and Dorlone, a selective nematocide[3]. Dowfume MC-2 was applied at the rate of 2 pounds per 100 square feet under a 4-mill thick polyethylene tarpaulin. Dorlone was applied with tractor mounted chisels at a depth of 6 inches and a spacing of 10 inches. After Dowfume MC-2 was applied the tarpaulin remained on the treated area for 48 hours. Following this a period of 3 days was allowed for soil aeration. After Dorlone was applied the treated areas were left for 2 weeks before planting. All of the areas treated in 1957 were 15 by 50 feet and were replicated three times in each field. The areas within each of the plots used for plant measurements and yield data were 7 by 30 feet.

In 1958, one field was fumigated with Dorlone at the rates of 16 and 24 gallons per acre and with Telone[4] at 16 and 32 gallons per acre. Because of the uniformity of both plant growth and nematode control at the two rates of application, the two rates were considered as two replicates of the same treatment. Both of the fumigants were applied with tractor mounted chisels, as in 1957. The size of the fumigated plots was 40 by 1100 feet, and the two areas within each of the fumigated plots utilized for sampling were 14 by 50 feet.

The effect of nematodes on the development of root rot and yield was also studied in the greenhouse. Soil was collected from the non-treated areas of the three fields fumigated in 1957. Dowfume MC-2 and Dorlone were applied to the soil at the same rate as used in the field studies, 2 pounds per 100 square feet and 25 gallons per acre, respectively. Each treatment in the greenhouse was replicated five times and four plants were grown per replicate. After treatment the soil was aerated for 2 weeks before planting.

Root rot severity was determined by the method described by Johnson (2). In the field, 200 plants at the bloom stage were examined for root rot severity in each of the treatments. In the greenhouse the severity of root rot was determined when the plants reached canning maturity. A variety of peas with wrinkled seed was used throughout these studies.

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[3] Dowfume MC-2--98% methyl bromide and 2% chloropicrin.
Dorlone--75% dichloropropene and 25% ethylene dibromide.

[4] Telone--technical dichloro propene.

The yield of peas in the field is expressed as the weight of shelled peas per acre, determined by sampling a representative area from each of the treated plots. The yield of peas in the greenhouse is expressed as the grams of pods, containing at least one pea of canning size per replication.

Soil from the field to be processed for nematode recovery was collected from ten sites within each sampling area from the rhizosphere of the pea plants. In the greenhouse, soil was collected from each of the replicates of each treatment. Nematode populations in the soil were determined by processing 500 cc of non-compacted soil by the method described by Christie and Perry (1), with final collection of the nematodes on a 270-mesh standard testing sieve. Further separation of nematodes and soil debris was accomplished with the Baerman Funnel technique. The number of parasitic nematodes in each soil sample was determined by counting their frequency of occurrence in a fraction of the total sample and calculating the total number based on this frequency.

EXPERIMENTAL RESULTS

Effect of Soil Fumigation on Root Rot

In all of the fumigation trials, Dowfume MC-2 controlled root rot, but Dorlone and Telone did not appreciably alter the severity of root rot (Table 1). In the 1957 field studies, the average root rot severity for the Dowfume MC-2 treated plots was 1.9. Plants grown on the Dorlone and non-treated areas had an average root rot severity of 3.1 and 3.8, respectively. The root rot severity of the plants grown in the field soil in the greenhouse paralleled very closely the results obtained in the field studies. The average root rot severity of plants grown in the Dowfume MC-2 and Dorlone treated soils was 1.6 and 5.7 and in the non-treated soil the average root rot severity was 6.7. Root rot was more severe in the greenhouse presumably because the plants were examined later than those in the field. Plants grown in the field, in 1958, in the soil treated with Dorlone and Telone had an average root rot severity of 2.3 and 2.1 and the plants grown on the non-treated soil had an average root rot severity of 2.4.

Table 1. Effect of soil fumigation on the severity of root rot of canning peas.

Year and location	Fumigant	Root rot severity[a]			
		Field A	Field B	Field C	Mean
1957 Field	Dowfume MC-2	1.8	2.0	2.2	2.0
	Dorlone	1.7	4.1	3.6	3.1
	Non-treated	2.2	4.5	4.9	3.9
1957 Greenhouse	Dowfume MC-2	2.7	1.3	1.1	1.7
	Dorlone	9.6	3.4	4.2	5.7
	Non-treated	9.9	4.8	5.4	6.7
1958 Field	Dorlone				2.3[b]
	Telone				2.1
	Non-treated				2.4

[a] Root rot severity based on a scale of 1 to 10. One represents a healthy plant and 10 a dead plant.
[b] Average of two replications from one field.

Effect of Soil Fumigation on Yield

The application of Dowfume MC-2, Dorlone and Telone to the soil increased the yield of shelled peas as indicated in Table 2. In 1957, plants grown on the Dowfume MC-2 and Dorlone treated soil averaged 2094 and 2190 pounds per acre of shelled peas, while the yield from the

Table 2. Effect of soil fumigation on the yield of canning peas.

Year and location	Fumigant	Yield of peas[a]			
		Field A	Field B	Field C	Mean
1957 Field	Dowfume MC-2[b]	1832	2283	2168	2094
	Dorlone	2109	2657	1804	2190
	Non-treated	1625	2194	1573	1797
1957-8 Green-house	Dowfume MC-2	15.4	22.8	23.5	20.6
	Dorlone	3.0	21.2	15.8	13.3
	Non-treated	0.6	16.2	6.1	7.6
1958 Field	Dorlone				6052[c]
	Telone				5896
	Non-treated				5492

[a] The yield from the field trials is expressed as pounds of shelled peas per acre, and in the greenhouse trials as the weight of green pods per replicate.
[b] The yields are low because of a toxic effect of the fumigant to the pea seedlings due to insufficient aeration period.
[c] Average of two replications from one field.

Table 3. Effect of fumigation on the number of parasitic nematodes recovered from the soil.

Year and location	Fumigant	Number of parasitic nematodes recovered[a]			
		Field A	Field B	Field C	Mean
1957 Field	Dowfume MC-2	64	16	1	27
	Dorlone	19	11	0	10
	Non-treated	664	42	7	238
1957 Green-house	Dowfume MC-2	0	0	0	0
	Dorlone	0	0	0	0
	Non-treated	330	222	62	205
1958 Field	Dorlone				68[b]
	Telone				30
	Non-treated				626

[a] Number of nematodes belonging to the following genera recovered from 500 cc of soil: Tylenchorhynchus, Paratylenchus, Xiphinema, Pratylenchus, Hoplolaimus, Trichodorus.
[b] Average of two replications from one field.

non-treated soil was 1748 pounds per acre. The average pod weight in grams from plants grown in the greenhouse on soils treated with Dowfume MC-2, Dorlone and non-treated was 20.6, 13.3, and 7.6, respectively. The average weight of shelled peas, in 1958, in the Dorlone and Telone treated plots was 6052 and 5896 pounds per acre, respectively, and from the non-treated areas 5528 pounds.

Effect of Soil Fumigation on Nematode Populations

In all tests the three fumigants were equally effective in the control of parasitic nematodes in the soil (Table 3). In these studies all of the nematodes belonging to the following genera were considered plant parasitic: Tylenchorhynchus, Paratylenchus, Xiphinema, Helicotylenchus, Hoplolaimus, Pratylenchus and Trichodorus. In the fields fumigated in 1957 an average of 238 parasitic nematodes were recovered from the non-treated soils, 27 from the Dowfume MC-2, and 10 from the Dorlone treated soils. In the soil fumigated in the greenhouse Dowfume MC-2 and Dorlone completely eradicated the parasitic nematodes, while in the non-treated soil an average of 205 parasitic nematodes were recovered from 500 cc of non-compacted soil. In 1958 the field soil treated with Dorlone and Telone had an average recoverable parasitic nematode population of 68 and 20, respectively, while in the non-treated soil 628 parasitic nematodes were recovered from 500 cc of non-compacted soil.

DISCUSSION

These results indicate that parasitic nematodes can reduce the yield of canning peas. However, the relationship of nematodes to the development of root rot was not established. The increase in yield resulting from the application of the selective nematocides may have been due to elimination of parasitic nematodes only. The small increase in severity of root rot in the areas not treated with the nematocides may be biologically significant, but further studies are necessary to establish the relationship of nematodes to the development of root rot.

These results demonstrate that plant parasitic nematodes are important in the root rot complex of canning peas in Minnesota, but whether they predispose the plant to root rot or are important parasites on peas in the absence of other root parasites has yet to be determined.

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EFFECTS OF A ROOT-KNOT NEMATODE MELOIDOGYNE INCOGNITA ACRITA CHITWOOD 1949 AND A STUBBY-ROOT NEMATODE TRICHODORUS CHRISTIEI ALLEN 1957 ON THE NUTRIENT STATUS OF TOMATO, LYCOPERSICON ESCULENTUM HORT. VAR. CHESAPEAKE[1]

M. O. Maung and W. R. Jenkins

Abstract

The effects of low and high infection levels of Meloidogyne incognita acrita and Trichodorus christiei on N, P, K, Na, Ca, Mg, reducing sugar, non-reducing sugar, and total sugar status of tomato tops and roots was determined. Low infection level of M. incognita acrita caused no significant change in percent constituents. There was a corresponding increase in all constituents when check plants had a higher dry weight than inoculated plants. However, a high level of infection caused an increased accumulation of N, P, and K in roots of infected plants. T. christiei had no effect on percent concentration of constituents, but there were marked differences in total accumulation corresponding to the weight differences.

INTRODUCTION

Many diseases are known in which host nutrition has an effect on the development and life cycle of the pathogen and on the degree of infection; or, conversely, the pathogen has an effect on the nutrient status of the host. Investigations have been made on plant diseases incited by bacteria, viruses, and fungi in an attempt to clarify the relationship between host nutrition and infection (1, 20, 22). Such investigations upon nematode diseases are limited.

The relationship between host nutrition and nematode infection can be determined in two ways: first, by direct determination of the nutrient status of the host under different levels of nematode attack; and second, by determining the rate of reproduction, development, and length of the life cycle of the parasite under different nutrient levels supplied to the host.

A study somewhat connected with nutrition of plants under attack by nematodes was made by Bessey (3), who conducted several fertilizer experiments with various plants growing in soil heavily infested with root-knot nematodes. He found that K exerted "a remarkable beneficial effect upon the plants" and enabled them to give a good crop despite the presence of nematodes. He further found that phosphatic fertilizer did not improve plant growth. Vanha (23) and Wilfarth and Wimmer (24) analyzed sugar beets infected with Heterodera schachtii and observed that diseased beets were characterized by low Ca, P, Mg, N, and very low K. They concluded that the beets were deprived of these elements due to the presence of nematodes. Roemer (15) was able to increase yield of sugar beets growing in H. schachtii infested soil from 18,000 to 19,000 kgms per 2 1/2 acres by applying K fertilizer alone. Hellinja (8), in his study with sugar beets infected with H. schachtii, observed that Ca and N concentrations of both tops and roots were higher while content of Mg, P, and K was lower than that of uninfected plants. He also found that the mineral composition varied according to the size of the nematode population. Solutions of various trace elements were applied to potato plants growing in soil naturally infested with H. rostochiensis by Ellenby (6) in an attempt to control potato sickness. He found that boric acid, $ZnSO_4$, and $MnCl_2$ gave increased yield. In was concluded that potato sickness would be more severe in soil deficient in B, Zn, and Mn. Tarjan (21) investigated roots of boxwood infected with Pratylenchus spp. and found that they contained higher levels of N and Na, while roots of checks contained higher levels of P. However, leaves of check plants had a higher level of K. An experiment to determine the nutritional disturbance in walnut brought about by P. vulnus under greenhouse conditions was carried out by Lownsbery (10). He observed that infected plants were deficient for K. It was therefore concluded that the symptoms of this disease resulted in part from K deficiency brought about by

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the nematodes' use of this element, their effect on the absorbing ability of the roots, or both. Sher (18) also observed K deficiency in roses infected with P. vulnus. Root-knot nematode infected lima beans were observed by Parris and Jehle (14) to be deficient in P despite an adequate supply of this element in the vicinity of the plants in the soil. Chitwood, Specht, and Havis (4) found changes in K, Ca, and Mg concentration in leaves of peach plants infected with root-knot nematodes. In an attempt to determine the source of N in relation to the development and rate of reproduction of M. incognita, Oteifa (13) observed that NH_4 had an inhibitory effect on the hatching of eggs and penetration of nematode larvae into roots. However, he found that neither NO_3, nor NH_4 supplied to plants affected the rate of reproduction and development of nematodes already within roots. The influence of N and K on the relationship between M. incognita acrita and soybeans was studied by Shands and Crittenden (17) who grew two varieties of soybeans supplied with three levels of K and N. They found that as the K and N supply was increased, penetration of the resistant variety by nematode larvae and the number of galls on the susceptible variety were increased. They concluded that the presence of nodule bacteria would increase the severity of root-knot disease. Oteifa (11, 12), in an experiment with M. incognita-infected lima beans growing in nutrient-supplied liquid medium, found that an increase in K concentration greatly increased the rate of reproduction and number of mature females. An experiment to determine the nutritional relationship between the root-knot nematode and its host plant was carried out by Dropkin (5) with tomato plants growing in a nutrient-supplied vermiculite medium. By using P^{32} in the nutrient solution supplied to the plants, and by measuring its accumulation in the galled roots, in uninfected roots, in individual nematodes, and in the tops of plants, he concluded the following: 1) P content of root-knot nematodes was fairly constant throughout their developmental stages and was negligible in the entire P content of the plants, 2) the unhealthy condition of plants was not due to robbing of the host by the nematodes of the vitial element, 3) galled roots absorbed this element at a slower rate than healthy roots in the same plant, and 4) less P was translocated out of the galled roots to the tops.

It was evident from the literature that additional information on this subject would be highly desirable. Therefore, investigations were begun to determine the effects of endoparasitic and ectoparasitic nematodes on the nutritional status of tomato.

MATERIALS AND METHODS

General Experimental Procedures

Four different experiments were performed with tomato, Lycopersicon esculentum Mill. hort. var. Chesapeake inoculated with either a low or high level of either Meloidogyne incognita acrita Chitwood 1947, or Trichodorus christiei Allen 1957. Each experiment consisted of 10 inoculated and 10 uninoculated plants in 4-inch-diameter clay pots arranged on a greenhouse bench in a completely randomized design. Supplemental light to supply a 15-hour day was provided by a series of 300 watt incandescent lamps placed at 4-foot intervals at a height of about 4 feet about soil level. The greenhouse was maintained at a night temperature of 65° to 70° F. Thirty days after each experiment was begun, all plants were fertilized equally with a complete fertilizer.

Preparation and Inoculation of Tomato Plants with Root-knot Nematodes

Specimens used as inoculum in these experiments were M. incognita acrita obtained from the Nematology Section, Plant Industry Station, Department of Agriculture, Beltsville, Maryland and maintained in greenhouse culture on tomato. To obtain material for use as inoculum, galls were collected from roots of this culture, washed thoroughly in running water, chopped up into small pieces, and triturated for 10 seconds in a Waring Blendor. The resulting mixture was then diluted with water.

Seeds of tomato were germinated in vermiculite. When seedlings were 18 days old, pots were two-thirds filled with steam sterilized soil and one or two teaspoonfuls of root-knot suspension were spread over the soil, depending upon whether inoculation was at a low or high level. One plant per pot was then placed directly over this layer and the pots finally filled with soil.

Preparation and Inoculation of Tomato Plants with Stubby-root Nematodes

T. christiei specimens were obtained from a population maintained in the University of Maryland greenhouse on tomato. To obtain inoculum, nematodes were extracted from soil by following a procedure which was a modification of the method described by Seinhorst (16). Nematodes were then collected individually under a stereoscopic microscope with the aid of a micropipette. A known number of nematodes was then placed in a known volume of water. Plants were transplanted singly into pots and inoculation was accomplished by pipetting aliquots of a nematode suspension containing either 600 or 2500 specimens into holes made in the soil near the base of the plant.

Analysis of Plant Materials

Sixty days after inoculation, plants were harvested in pairs to obtain sufficient material for analysis. After washing the roots in running water, tops and roots of each pair of plants in each experiment were cut into pieces and dried in an oven at 75° to 85° C for 36 hours. In the case of plants inoculated with M. incognita acrita, a root-knot index was determined by following the procedure described by Smith and Taylor (19).

After drying, tops and roots of each pair of plants were weighed on a triple beam balance and their weights recorded. All dry materials were separately ground in a Wiley Mill through a 40-mesh sieve and stored in air tight bottles. Concentrations of N, P, K, Na, Ca, Mg, reducing sugar, and total sugar were determined in both tops and roots of each pair of plants by the following methods. Total N was analyzed for by following the procedure described by Linder (9) using a photoelectric colorimeter equipped with 425μ filter. P and Mg were determined by following the procedure described in AOAC (2) using the same colorimeter equipped with 640μ and 525μ filters, respectively. K, Na, and Ca were also determined by the colorimetric method using a Beckman Model DU spectrophotometer with a flame attachment (2). Reducing sugar and total sugar were analyzed for by following the Shaffer-Somogyi method as adapted by Heinze and Murneek (7). Non-reducing sugar was obtained by subtracting the value of reducing sugar from that of total sugar. For the determination of water soluble N, a known weight of plant material was soaked in a known volume of distilled water for 1 hour with an occasional thorough shaking. After filtration, N content in an aliquot of filtrate was determined as described above. The total N content in the entire plant material was then calculated mathematically.

RESULTS AND DISCUSSION

Effects of M. incognita acrita on the Growth and Nutrient Status of Tomato at a Low Infection Level

The root-knot index of these plants, determined 60 days after inoculation, showed 25 to 50 percent of the roots galled. This amount of infection significantly reduced growth of tops from a mean dry weight of 19.8 gm to 18.0 gm per two plants. The dry weight of roots increased from 8.1 gm to 8.9 gm, a non-significant difference. Effects of this infection level on the nutrient status of the host plants are summarized in Table 1. N, P, K, Ca, and Mg concentrations in tops of check plants were slightly higher than those in the inoculated plants. Except for Mg and Ca the total and percent concentration of all the above elements were slightly higher in roots of infected plants, but differences were not significant. However, total accumulation and percent Na concentration in the check plants was significantly higher in both tops and roots. No difference was found in sugar content between check and inoculated plants.

Effects of M. incognita acrita on Growth and Nutrient Status of Tomato at a High Infection Level

The root-knot index of these plants, determined 60 days after inoculation, showed that roots were 51 to 75 percent galled. This infection significantly reduced the growth of tops and root systems from the mean dry weights of 22.0 gm to 12.7 gm and 9.2 gm to 7.2 gm per two plants, respectively. Effects of nematodes at this infection level on the nutrient status of tomato are shown in Table 2. The total accumulation and percent concentration of N, P, K, Na, Ca, and Mg in tops of check plants was higher than that in tops of inoculated plants, but differences were not significant.

Table 1. Effects of Meloidogyne incognita acrita Chitwood 1949 on the nutrient status of tomato, Lycopersicon esculentum Mill. hort. var. Chesapeake having a root-knot index of 26 to 50 per cent 60 days after inoculation. Values are expressed as a mean of five replicates.

Constituents	Totals in milligrams and as per cent of dry weight per 2 plants			
	Check		Inoculated	
	Total	Per cent	Total	Per cent
	TOPS			
N	378.20	1.89	288.00	1.60
P	120.80	0.61	102.60	0.57
K	530.60	2.68	460.80	2.56
Na	110.80	0.56	72.00	0.40*
Ca	932.50	4.71	784.80	4.36
Mg	87.10	0.44	72.00	0.40
Reducing Sugar	23.80	0.12	19.80	0.11
Non-reducing Sugar	156.40	0.79	138.60	0.77
Total Sugar	180.20	0.91	158.40	0.88
	ROOTS			
N	143.40	1.77	176.40	1.96
P	38.10	0.47	46.30	0.52
K	63.20	0.78	71.20	0.80
Na	32.40	0.40	29.40	0.33*
Ca	22.70	0.28	23.10	0.26
Mg	40.50	0.50	37.40	0.42
Reducing Sugar	9.70	0.12	97.90	0.11
Non-reducing Sugar	59.10	0.73	65.90	0.73
Total Sugar	68.85	0.85	74.80	0.84

* Denotes significance at the 5 per cent level.

Table 2. Effects of Meloidogyne incognita acrita Chitwood 1949 on the nutrient status of tomato, Lycopersicon esculentum Mill. hort. var. Chesapeake, having a root-knot index of 51 to 75 per cent, 60 days after inoculation. Values are expressed as a mean of five replicates.

Constituents	Totals in milligrams and as per cent of dry weight per 2 plants			
	Check		Inoculated	
	Total	Per cent	Total	Per cent
N	332.20	1.51	167.60	1.32
Water Soluble N	18.50	0.084	8.60	0.068
P	103.40	0.47	55.90	0.44
K	462.00	2.10	215.90	1.70
Na	55.00	0.25	26.70	0.21
Ca	541.20	2.46	298.50	2.35
Mg	107.80	0.49	54.60	0.43
Reducing Sugar	24.20	0.11	15.20	0.12
Non-reducing Sugar	156.20	0.71	86.40	0.68
Total Sugar	176.00	0.80	101.60	0.80
	ROOTS			
N	157.30	1.71	244.10	3.39**
Water Soluble N	12.30	0.134	5.50	0.077
P	41.40	0.45	43.90	0.61*
K	72.70	0.79	180.00	2.50*
Na	80.00	0.87	20.80	0.29*
Ca	43.20	0.47	31.00	0.43
Mg	64.30	0.68	25.90	0.36
Reducing Sugar	10.10	0.11	7.90	0.11
Non-reducing Sugar	65.30	0.71	51.10	0.71
Total Sugar	75.40	0.82	59.00	0.82

* Denotes significance at the 5 per cent level

** Denotes significance at the 1 per cent level

Table 3. Effects of Trichodorus christiei Allen 1957 on the nutrient status of tomato, Lycopersicon esculentum Mill. hort. var. Chesapeake, 60 days after inoculation with 600 nematodes per plant. Values are expressed as a mean of five replicates.

Constituents	Totals in milligrams and as per cent of dry weight per 2 plants			
	Check		Inoculated	
	Total	Per cent	Total	Per cent
	TOPS			
N	693.00	3.15	516.10	2.78
P	165.00	0.75	140.30	0.75
K	682.00	3.1	561.00	3.00
Na	46.20	0.21	33.70	0.18
Ca	536.60	2.43	417.00	2.23
Mg	101.20	0.46	82.30	0.44
Reducing Sugar	24.20	0.11	20.60	0.11
Non-reducing Sugar	165.00	0.75	136.50	0.73
Total Sugar	189.20	0.86	157.10	0.84
	ROOTS			
N	258.30	2.46	182.50	2.31
P	67.20	0.64	48.20	0.61
K	51.50	0.49	36.30	0.46
Na	33.60	0.32	19.80	0.25
Ca	15.80	0.15	11.90	0.15
Mg	59.90	0.57	45.00	0.57
Reducing Sugar	8.40	0.08	6.30	0.08
Non-reducing Sugar	67.20	0.64	50.60	0.64
Total Sugar	75.60	0.72	56.90	0.72

Table 4. Effects of Trichodorus christiei Allen 1957 on the nutrient status of tomato, Lycopersicon esculentum Mill. hort. var. Chesapeake, 60 days after inoculation with 2500 nematodes per plant. Values are expressed as a mean of five replicates.

Constituents	Totals in milligrams and as per cent dry weight per 2 plants			
	Check		Inoculated	
	Total	Per cent	Total	Per cent
	TOPS			
N	472.00	2.36	259.90	2.13
P	150.00	0.75	79.30	0.65
K	680.00	3.40	353.80	2.9
Na	42.00	0.21	22.00	0.18
Ca	494.00	2.47	279.40	2.29
Mg	80.00	0.40	41.50	0.34
Reducing Sugar	24.00	0.12	14.60	0.12
Non-reducing Sugar	148.00	0.74	89.10	0.73
Total Sugar	172.00	0.86	103.70	0.85
	* *		ROOTS	
N	247.80	2.95	153.90	2.70
P	57.90	0.63	38.20	0.60
K	27.70	0.33	14.80	0.26
Na	18.50	0.22	9.70	0.17
Ca	21.00	0.25	12.50	0.22
Mg	47.90	0.57	38.20	0.67
Reducing Sugar	9.20	0.11	5.70	0.10
Non-reducing Sugar	63.80	0.76	41.00	0.72
Total Sugar	73.10	0.87	47.30	0.83

Percent N, P, and K concentrations in roots of inoculated plants were significantly higher than those in roots of check plants. Percent N concentration in roots of inoculated plants was significantly higher than that in roots of check plants. There was also an increase in total accumulation of N in inoculated plant roots as compared with the roots of check plants, 244.1 mg to 157.3 mg per two plants. However, the total accumulation of water soluble N in the roots of check plants was more than twice as much as that in the roots of infected plants, 12.30 mg to 5.50 mg per two plants, indicating that the high N accumulation in the infected roots was due to protein. Both total accumulation and percent concentration of K in roots of infected plants were greater in comparison with the roots of check plants. Little difference was observed in total P accumulation, inoculated plant roots having 43.9 mg to 41.4 mg for check plant roots. This is in accordance with Dropkin (5) who reported that the P content of an individual nematode at any stage of its development was constant and was negligible for the total accumulation in the plant.

Effects of T. christiei on the Growth and Nutrient Status of Tomato Following Inoculation with 600 Nematodes per Plant

Growth of tops of inoculated plants was reduced from a mean dry weight of 22.0 gm to 18.7 gm and of roots from 10.5 gm to 7.9 gm per two plants. As shown in Table 3, no difference in composition between check and inoculated plants was observed except for a slightly greater total accumulation and slightly higher percent concentration of most nutrients in roots and tops of check plants.

Effects of T. christiei on the Growth and Nutrient Status of Tomato Following Inoculation with 2500 Nematodes per Plant

This infection caused a highly significant reduction in growth of tops from a mean dry weight of 20.0 gm to 12.2 gm and of roots from 8.4 gm to 5.7 gm. Effects of nematodes on the nutrient status of plants are shown in Table 4. There was a slightly higher percent concentration of mineral constituents in checks, which was not statistically significant. No difference was observed in sugar composition. As a result of the great difference in dry weight, there was a corresponding increase in total accumulation of constituents in check tops and roots, compared with inoculated tops and roots. It was obvious from the highly significant difference in dry weights between check and inoculated plants that plants were severely injured. These injuries have been characterized by reduced yield, low vigor, general decline, and typical dwarfing, but not by symptoms usually associated with mineral deficiencies. These effects might be caused not by the effect on the nutrition of plants, but by the reduction of root surface, thereby reducing the total amount of absorption by the roots. There was no evidence of accumulation of nutrients in the roots resulting from interference with upward translocation, as there was with root-knot nematodes.

As far as sugar was concerned, neither M. incognita acrita nor T. christiei affected concentrations of these compounds in plants.

SUMMARY AND CONCLUSION

M. incognita acrita, an endoparasitic nematode, did not have a significant effect on the nutritional status of plants having a root-knot index of 25 to 50 percent, 60 days after inoculation, although there was a slightly higher accumulation of constituents in the roots of infected plants with the exception of Na and Mg. However, when the infection was increased so that the infected plants had a root-knot index of 51 to 75 percent, there was a significantly higher level of percent concentration and total accumulation of N and K. On the contrary, though percent concentration of P in the infected plant roots was significantly higher, the total accumulation did not show a significant difference.

T. christiei, an ectoparasitic nematode, did not affect nutrient concentration, either at a low or high infection level, but it caused a decrease in the total accumulation of plant constituents through the reduction of tops and roots by the formation of short stubby roots.

In general, it was concluded that: 1) plants highly infected with root-knot nematodes accumulated N, P, and K in high amounts in the roots, 2) either the absorption for Na by the roots was prevented or a great portion of Na was lost by the roots due to the effect of nematodes, 3) T. christiei, either at the low or high infection level, did not affect the composition of the constituents of plants, but affected their total accumulation in the plants through the reduction

of tops and roots, 4) neither M. incognita acrita nor T. christiei affected the percent sugar content regardless of the rate of population.

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A ROOT-LESION NEMATODE DISEASE OF AVOCADOS

S. A. Sher[1], F. J. Foote[2], and S. B. Boswell[1]

Summary

Pratylenchus vulnus caused a stunting of avocado plants and a reduction in root growth in a greenhouse test. Similar symptoms have been seen in the field. Preplant soil fumigation resulted in marked increases in tree growth. Root-lesion nematode disease of avocados has only been seen where avocados have been planted after walnuts infested with *P. vulnus*.

Several avocado plantings in Ventura County in southern California, planted after removal of walnuts, have not produced satisfactory trees. Soil and avocado root samples have contained the root-lesion nematode, *Pratylenchus vulnus*. Trees were stunted, feeder roots were scarce, and the trees appeared more susceptible to frost injury. The root-lesion nematode is known to be an important pest of walnuts and other crops in California (1, 2), and therefore greenhouse and field fumigation tests were undertaken to investigate the relationship of *P. vulnus* to the growth of avocados.

GREENHOUSE INFESTATION TEST

Twenty-one avocado seedlings (*Persea americana* var. Mexicola) were planted in 10-inch clay pots in steam sterilized sandy loam. Two weeks after planting, seven pots were infested with 100 *Pratylenchus vulnus* per pot, seven pots with approximately 1000 per pot, and the remaining seven pots received no nematodes. The nematodes were obtained from soil around roses at Ontario, California. Pots were put in randomized blocks on a greenhouse bench.

One year after infestation, two 50-cc soil samples were taken from each pot and put on a modified Baermann funnel for 3 days and the *P. vulnus* counted. Two-gram root samples were put on a modified Baermann funnel for 3 days and the nematodes counted. Dry weights of the plants were taken.

RESULTS

There was a marked reduction of top growth in all the plants infested with nematodes (Fig. 1). Root systems were similarly reduced in growth in the two series infested with root-lesion nematode. *Pratylenchus vulnus* was recovered from all the soil and root samples in all the pots that were infested (Table 1). Dry weights of the plants in the uninfested series were highly significant over the two infested series (Table 1).

Table 1. Dry weights of avocado plants and *P. vulnus* recovered in the soil and roots.

Treatment	Dry weight of plants (in grams)	Number of nematodes per 100 cc soil	Number of nematodes per 2 gm roots
100 P. vulnus	54.8	183.9	63.4
1000 P. vulnus	38.4	159.6	47.3
Check	202.3	0	0

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FIGURE 1. Avocado greenhouse test. From left to right, Check, 100 P. vulnus, 1,000 P. vulnus.

FIGURE 2. Three-year-old avocado in the field. Grown in unfumigated soil at left, fumigated soil at right.

Table 2. Nematode counts at three soil depths for 2 years and tree measurements the third year after start of test.

Treatment		Number of nematodes per 100 cc of soil (soil depth in feet)			Height of tree (cm)	Width of tree (cm)
		0-1	1-2	2-3		
120 gallons of D-D per acre	1958	0	0	0.1	---	---
120 gallons of D-D per acre	1959	0	0	0.4	178.5	143.8
No treatment	1958	20.3	28.7	28.8	---	---
No treatment	1959	5.9	27.7	26.0	107.8	81.2

PREPLANT SOIL FUMIGATION

The root-lesion nematode infested soil in a former walnut orchard, removed in 1955, was fumigated in part in February 1957. D-D (1,3 dichloropropene-dichloropropane mixture) at 120 gallons per acre was applied by chisel application at a depth of 14 inches. Avocados (var. Zutano) were planted 3 months after fumigation.

One year after fumigation soil samples were taken from around 15 trees in treated soil and 15 trees in untreated soil. Four soil samples at 1-foot depths to 3 feet were taken from each tree and each level from each tree was composited. Two 50-cc soil samples were put on a modified Baermann funnel for 3 days and the P. vulnus counted (Table 2). Fumigated tree sites had trees that were approximately twice the size of unfumigated sites.

The second year after fumigation similar soil samples were taken and recorded (Table 2). Tree measurements were taken at this time (Table 2). Trees in the treated sites continued to show marked growth increases over the untreated tree sites (Fig. 2).

DISCUSSION

Results of these tests indicate that Pratylenchus vulnus can cause extensive damage to young avocado trees. Preplant fumigation with D-D is indicated when this nematode is present in land to be planted to avocados. Walnut orchards in California are usually infested with P. vulnus, and the planting of avocados after walnuts would probably result in root-lesion nematode injury to avocados.

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THE PINE CYSTOID NEMATODE IN NEW JERSEY

M. T. Hutchinson and J. P. Reed

In July 1956, endoparasitic nematodes were found in roots taken from the vicinity of a wild highbush blueberry, Vaccinium corymbosum, growing at the intersection of the Oswego and Wading rivers near Harrisville, New Jersey, 7 miles north of New Gretna.

These nematodes were not initially identifiable, but after resampling of the same blueberry bush on June 2, 1958 they were found to be associated with a considerable number of globular females (Fig. 1).

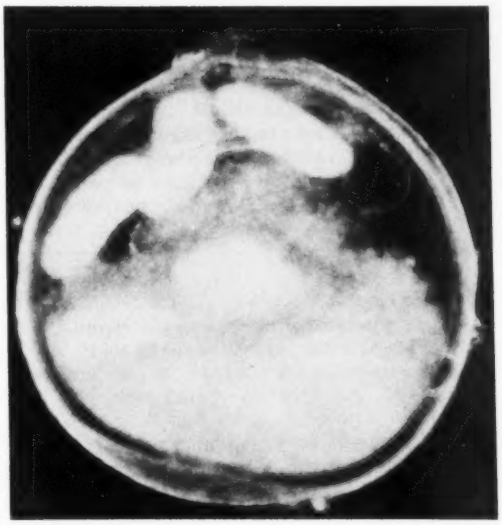

FIGURE 1. Female of Meloidodera floridensis, containing several eggs.

These females and larvae were identified as Meloidodera floridensis at the USDA Nematology Section, Beltsville, Maryland.

After this identification, a more careful sampling of the Harrisville location on August 20 showed that the Meloidodera larvae were present in numbers up to 2200 per gram inside the roots of a specimen of pitch pine, Pinus rigida[1], but were not present inside the roots of the aforesaid blueberry bush with which the pine roots were entangled. Later, on November 25, sampling showed moderate numbers of larvae in the roots of the Pinus rigida, and a few larvae in the roots of an adjacent specimen of short-leaf pine, Pinus echinata[1].

Samplings of P. rigida at other areas on November 25 showed a few Meloidodera larvae to be present in roots at locations near Toms River, and 5 miles south of Chatsworth, respectively. No larvae or females were present at locations near Old Bridge and New Lisbon.

Occasional larvae have also been found around the roots of wild blueberry, Vaccinium corymbosum and V. vacillans, at locations 5 miles south of Toms River and 3 miles south of Smithsburg, respectively.

Measurements of living and preserved larvae from two New Jersey locations fall mostly below the size range given by Chitwood et al. (1). The type population is stated to range from 500 to 559 μ in length while the New Jersey populations (from Harrisville and Toms River) ranged from 462 to 550 μ, and from 394 to 542 μ, respectively. A total of 55 specimens was measured.

The results of this preliminary survey suggest that Meloidodera floridensis is native to New Jersey and, as in the southern states, is a parasite of pine (1, 2). Pinus rigida and possibly P. echinata are new host records.

[1] Identifications confirmed by Dr. Murray Buell, Department of Botany, Rutgers University.

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FUSICOCCUM CANKER OF THE HIGHBUSH BLUEBERRY IN MASSACHUSETTS[1]

Bert M. Zuckerman[2]

Fusicoccum canker, caused by Fusicoccum putrefaciens Shear, was first found in Massachusetts in 1957. Severe outbreaks occurred in several fields in 1958, and in 1959 the disease appears to be more widespread. To date, the disease has been found in eight fields and has been identified from the Rubel, Jersey, Pioneer and Cabot varieties of the cultivated highbush blueberry, Vaccinium corymbosum L.

In 1958, McKeen (2) described the disease syndrome as it occurs in British Columbia, and reported on investigations which established the pathogenicity of F. putrefaciens on the highbush blueberry. Concurrently, Creelman (1) published a similar paper which related to investigations of this disease in Nova Scotia. Creelman (1) states that Fusicoccum canker has been found in Maine from specimens collected by Dr. M. T. Hilborn, but does not indicate how widespread the disease is in this area.

In Eastern Massachusetts, sporulating pycnidia were found in abundance from the first of March through mid-July. Creelman (1) states that pycnidia appear in dead bark in cankers for about 5 weeks, beginning in late July, in Nova Scotia. These variations in the time and duration of pycnidial formation probably are due to differences in climate in Eastern Massachusetts as compared with Nova Scotia. McKeen (2) reported that pycnidial initials form in early March in British Columbia.

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CALYX-END ROT OF APPLES[1]

J. F. Hockey[2]

Abstract

A calyx-end rot of apples caused by Sclerotinia sclerotiorum is present in sod mulch orchards in Nova Scotia. Sclerotia are produced on the affected fruits after they fall to the ground. These sclerotia produce apothecia and ascospores the following spring. The varieties McIntosh and Red Rome Beauty have been the most seriously affected.

INTRODUCTION

Sclerotinia sclerotiorum (Lib.) d By. was reported by Hockey (2, 3) to cause a calyx-end rot of apples in certain orchards in Nova Scotia. It has been found affecting 2 percent of the fruit of McIntosh and Linda and 12 percent of the variety Red Rome Beauty. The fungus has been previously reported as causing a storage rot of apples and pears in New Zealand (1) and a blossom blight and green fruit rot of apricots in California (4). The fungus is of common occurrence in Nova Scotia and has caused severe losses in various vegetable and forage crops on many occasions. This paper records some observations on this organism as it affects apples in the orchard.

This same disease or a similar disease, dry eye rot of apples, caused by Botrytis cinerea Pers., has been reported from England and New Zealand by Wilkinson (5). He described dry eye rot of apples in England as appearing in three stages "viz. 1) the formation of the original rot, 2) the quiescent Dry Rot, and 3) the development of the disease from stage 2) into one involving complete rot of the whole fruit." The three stages apply equally well to the progressive symptoms of calyx-end rot as observed in Nova Scotia.

SYMPTOMS

The first symptoms of the rot appeared soon after bloom; dark brown lesions appeared in the calyx basin of the apples. These lesions were found either laterally at the base of sepals or encircling the calyx. They also expanded slightly, during the early part of the summer, to the second stage described by Wilkinson and attained a diameter of 1 to 2 cm. At this time they were, in reality, small pockets of quiescent rot. The rotted tissue had desiccated leaving in the apple a cavity protected externally by a leathery cover formed from the epidermis and part of the parenchyma tissue. The center of the lesion surface was grayish-tan and was surrounded by a reddish border (Figs. 1 and 2).

Two to four weeks before harvest some fruits had an active brown rot extending from the older lesions and corresponding to Wilkinson's third stage. When such fruits were placed in moist chambers at room temperature or when they dropped to the orchard floor, the fungus permeated the entire fruit and produced sclerotia on the surface of the fruit at any break in the epidermis. Fruits that were in stages 1 or 2 of the rot when harvested have seldom developed the progressive rot in storage.

OBSERVATIONS

Samples taken from the margins of lesions and plated on potato-dextrose agar gave rise to mycelium and sclerotia of Sclerotinia sclerotiorum. Isolations made during the past 3 years, from fruits showing any of the described symptoms, have all yielded this fungus.

During the spring of 1958 a search was made for apothecia of the fungus in orchards where rot had been found the previous season. Apothecia were quite prevalent in the orchard mulch just before bloom, on May 23, and were sporulating freely by early June. Occasional sclerotia were found attached to the end of a 1957 fruitlet. Clouds of ascospores were observed to arise when the mulch was disturbed. Apothecia were still found under the mulch 3 weeks after bloom, when drier weather inhibited further development. The apothecia were present in all grass-

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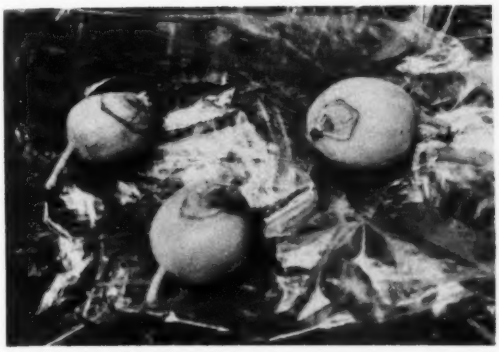

FIGURE 1. Young McIntosh fruits showing early symptoms of calyx-end rot.

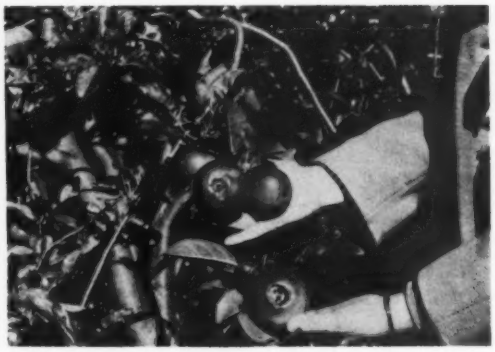

FIGURE 2. Fruits of Red Rome Beauty with quiescent rot symptoms prior to harvest.

mulch orchards in which the disease had been reported previously, but they have not been found in an orchard under clean cultivation.

Some blossom clusters were inoculated with a suspension of macerated apothecia just as the petals were falling, but failed to reproduce the disease.

Examination of the fruit on the orchard floor after harvest revealed the presence of sclerotia on the surface as well as in the cavities of decaying fruit.

DISCUSSION

The symptoms of this disease indicate that infection of the calyx end takes place through the withered petals that have remained adhering to the fruit and through the sepals. This period coincides with the greatest abundance of apothecia and spores. Infection must come from ascospores because the only other method of infection is direct contact with the mycelium. Since affected fruits have not been found in contact with the ground, the mycelium plays no part in causing infection.

The sclerotia fall in the mulch and after overwintering there produce apothecia in the spring. Smith (4) found that in apricot orchards the source of inoculum was not confined to the ground area but that spores could drift from some distance. The initial source of infection in apple orchards has undoubtedly been from sclerotia arising from infected hosts of the fungus or the favorable environment of supplemental mulches or manure applied to these orchards.

Although no experimental results are available on the use of fungicide sprays to control this disease, it would seem logical to apply a fungicide at the time the apothecia mature and thereby prevent infection from taking place. The normal spray schedule for the control of apple scab may not give adequate control of calyx-end rot in seasons when a prolonged infection period exists between the late pink and calyx applications. A special application of fungicide during the late bloom period may be necessary in seasons when weather conditions favor the production and maturity of apothecia.

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A PRELIMINARY NOTE ON THE CAUSE OF "PECKY" CYPRESS

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Summary

The principal decay in baldcypress is a well known disease in the south. The type of rot is unique and referred to commercially as "pecky" cypress. The cause of this decay has been the subject of some speculation among pathologists but has not been definitely determined. Cultures from spores of a Stereum sp., fruiting on a standing tree, were obtained and have the same characteristics as cultures isolated from numerous trees having the typical type of decay.

The first comprehensive account of "pecky" cypress, the principal decay disease of baldcypress (Taxodium distichum), was made by von Schrenk (4) in 1900. He showed that the disease was widespread in the upper parts of the trunk in older baldcypress. He did not identify the rot fungus, but believed that it entered the tree through branch stubs or wounds. Although most of the extensive older stands of cypress have been cut over, pecky cypress is still common.

Long (2) made an extensive survey of decay fungi on living cypress trees and concluded that Fomes geotropus was the probable cause of peckiness. Although Long stated that in some of the cypress trees on which F. geotropus was fruiting there was also the pecky type of rot, he did not show that there was a consistent and close association of the two. Murrill (3) also reported finding F. geotropus fruiting on cypress but gave no convincing proof that it caused the pecky decay. F. geotropus fruits mainly on or near the base of the trunks and is often associated with a hollow condition. It is found on several hardwood species (2, 3) where the decay is of the white type. F. geotropus on magnolia (1) has been described in detail.

In November 1958 one of the present authors collected a Stereum sp. on the main stem, at branch stubs, of baldcypress. Basidiospore cultures were obtained from this Stereum. Later a similar culture was obtained from decay at the base of the stub on which the fruiting developed. Two cultures obtained previously from pecky cypress decay, from Florida and Mississippi, were also similar.

In January 1959 pecky cypress decay specimens were obtained from two lumber mills where cypress logs were being cut. At Valdosta, Georgia[4], samples were obtained from eight trees, six of which yielded the same fungus in culture. Three additional samples were obtained at Jesup, Georgia[4], and two of these yielded the same fungus. No decay fungi were cultured from the other trees.

Later, pecky cypress boards from two trees were sent to Beltsville by C. S. Hodges from a mill near Elizabethtown, North Carolina. The same decay fungus was isolated. The fungus was readily isolated from all areas of the infected heartwood, from the advanced stages in the pockets of decay, from firm wood between the pockets, and from firm heartwood just inside the sapwood. Information on samples and isolations is given in Table 1.

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[3]Officer in Charge, Athens-Macon Research Center, Southeastern Forest Experiment Station, Forest Service, United States Department of Agriculture.

[4]The cooperation of the Langdale Lumber Company, Valdosta, Georgia and the Pioneer Wood Products Mill, Jesup, Georgia was very helpful and made possible the obtaining of more extensive information on this decay disease.

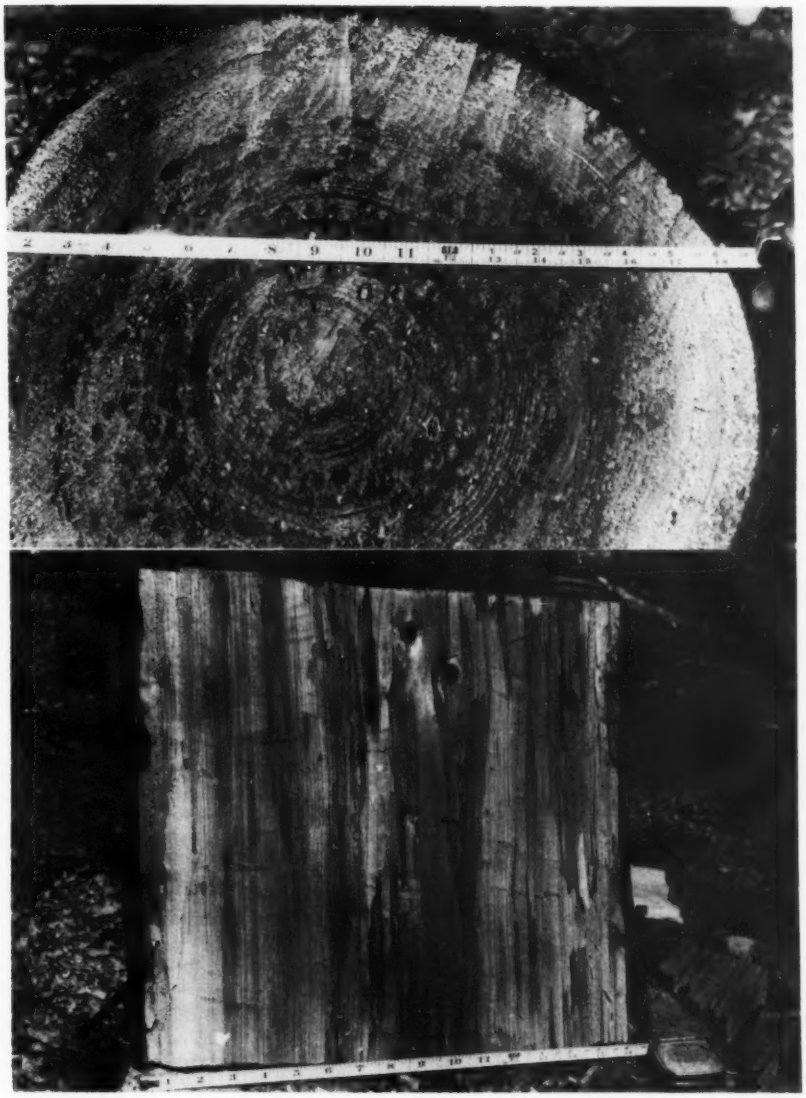

FIGURE 1. Above, a cross section through a log from an old cypress tree showing typical peck. Below, a vertical section through the same log.

Table 1. Information on Stereum cultures obtained from decayed cypress.

Locality	Date collected	Number trees sampled	Source of cultures	Condition	Collectors
Lake City, Fla.	Dec., 1945	1	Pecky decay	Lumber at mill	R. P. True
Stoneville, Miss.	July, 1953	1	Pecky decay	Cut tree	E. R. Toole
Stoneville, Miss.	Nov., 1958	1	Sporophore and decay	Standing tree	E. R. Toole
Valdosta, Ga.	Jan., 1959	6	Pecky decay	Lumber and logs	Otis Maloy, W. A. Campbell, R. W. Davidson
Jesup, Ga.	Jan., 1959	2	Pecky decay	Lumber and log	W. A. Campbell, R. W. Davidson
Elizabethtown, N.C.	Feb., 1959	2	Pecky decay	Lumber at mill	Charles Hodges
Stoneville, Miss.[a]	Mar., 1959	1	Pecky decay	Cut tree	E. R. Toole

[a]The tree from which this sample was taken had been dead about 1 year.

All decay specimens except the specimen from the standing tree at Stoneville, Mississippi (1958) had the typical pecky decay (Fig. 1). There were pockets of advanced decay scattered throughout the heartwood. The pockets in specimens with less advanced decay were whitish and the wood somewhat stringy or laminated, but the contents of the pockets in the more advanced stages of decay were mostly reduced to powder or were easily pulverized between the fingers. The decay is regarded as not typically a brown rot but intermediate between a white and brown rot. The tree on which the Stereum sp. was collected has not been cut or sampled sufficiently to determine the type of associated decay.

A detailed study of cultures of the cypress fungus is now under way. It will be identified, described, and compared with cultures and sporophores of related species. It seems probable that this research will lead to solution of the cause of pecky cypress.

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DROUGHT DAMAGE TO WESTERN WHITE PINE AND ASSOCIATED TREE SPECIES

Charles D. Leaphart[1]

Abstract

An unusually dry summer in 1958, characterized by abnormally high temperatures, damaged western white pine, western larch, grand fir, and lodgepole pine in an area in northeastern Washington and northern Idaho. Damage ranged from foliage wilting to death of trees, and affected trees from 3 to 25 feet tall.

The 1958 growing season in the Inland Empire was quite dry and considerably hotter than normal. These conditions, especially on severe sites, accounted for summer drought damage reported for the first time in the western white pine type. Western white pine (Pinus monticola Dougl.), western larch (Larix occidentalis Nutt.), grand fir (Abies grandis (Dougl.) Lindl.), and lodgepole pine (P. contorta Dougl.) suffered more severe injury than other species. However, damage to Douglas fir (Pseudotsuga menziesii var. glauca (Beissn.) Franco) and ponderosa pine (P. ponderosa Laws.) was also reported. Damage most often occurred to seedlings and small saplings, but some small pole-size trees were also injured.

Drought injury to trees is any damage caused by moisture deficiencies. It occurs primarily when the soil cannot supply the tree's demands for moisture. Drought injury symptoms vary but can occur as foliage wilting, foliage discoloration or death, reduced growth, twig dieback, or tree death. These symptoms may occur singly or in combination, depending on the intensity of damage.

Drought, essentially, affects the more succulent tissues of a tree first. When dieback occurs, the tree dies from the tip down and from the outside in. Discoloration of foliage is the most striking symptom of drought.

Nothing has been published on drought damage to conifers in the western white pine type. However, it is conceivable that this kind of injury did occur at various times in the past but was undetected, not recognized as such, or at least unreported.

Soil moisture apparently had neared the critical levels of availability by August 4, 1958 in the Priest River Valley in northern Idaho. On certain severe sites western larch started to turn yellow; some trees already had shed a portion of their foliage. Larch normally turns yellow late in the fall and often does not lose its needles until after the first snows.

A report was received in late August that some severe top damage of unknown origin occurred on planted white pine in the general area where larch had discolored earlier. This plantation is on State land approximately 1 mile north of McAbee Falls bridge along the Priest River. In addition to the planted white pine, other species that regenerated naturally within the plantation area are Douglas fir, grand fir, lodgepole pine, ponderosa pine, and western larch.

When the plantation was examined in September, the symptoms shown by affected western white pine trees were characteristic of sudden drought damage. Although some trees still had a normal green color then, the foliage of others was often distorted or drooping around the stem or branches (Figure 1). The leader and upper crown branches most commonly showed these symptoms. On other trees, where damage was slightly more severe, foliage had completely drooped around the branches or leaders and had usually turned a light straw color. Later this straw-colored foliage turned still darker and was reddish brown by mid-October. On all these trees, the uppermost and outermost portions of the crown were more severely affected than the rest of the crown (Figure 2). The more severe the drought effect was on an individual tree, the more the crown was discolored. Some trees were killed to the ground line (Figures 1, 2). The roots of all trees still appeared healthy in mid-October.

Most of the damaged trees in the plantation were western white pine. The damaged white pine in this area ranged from 3 feet to approximately 25 feet tall, but intensity of injury appeared to depend upon tree location rather than upon size. All trees had been planted in 1941. More than 60 percent of the planted trees showed some degree of damage (Figure 3).

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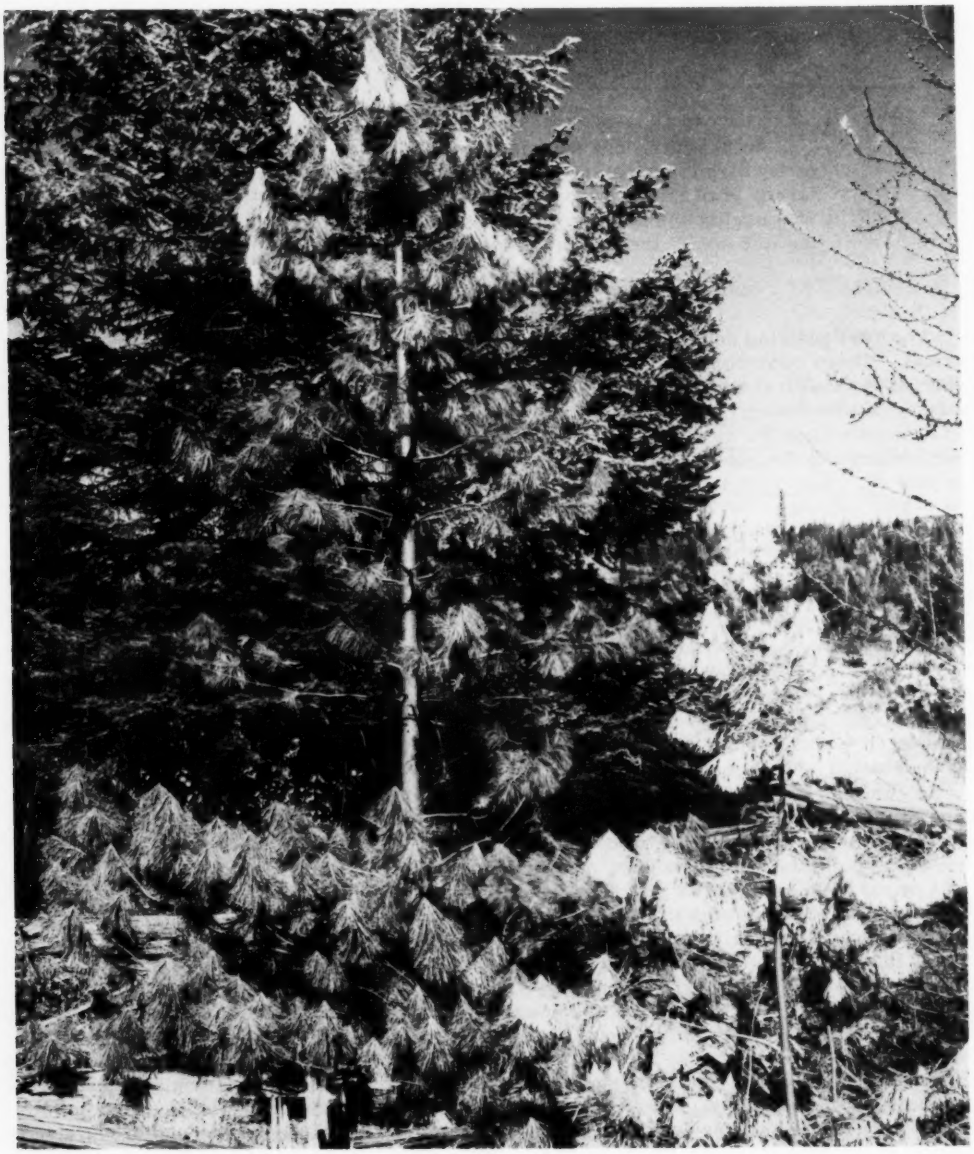

FIGURE 1. Two white pine trees showing different degrees of drought injury. The small tree at the right is dead to the ground line and shows the dead (white) foliage. The larger tree shows some needle mortality on the tips of upper crown branches. The needles in the lower crown show the drooping appearance characteristic of drought injury.

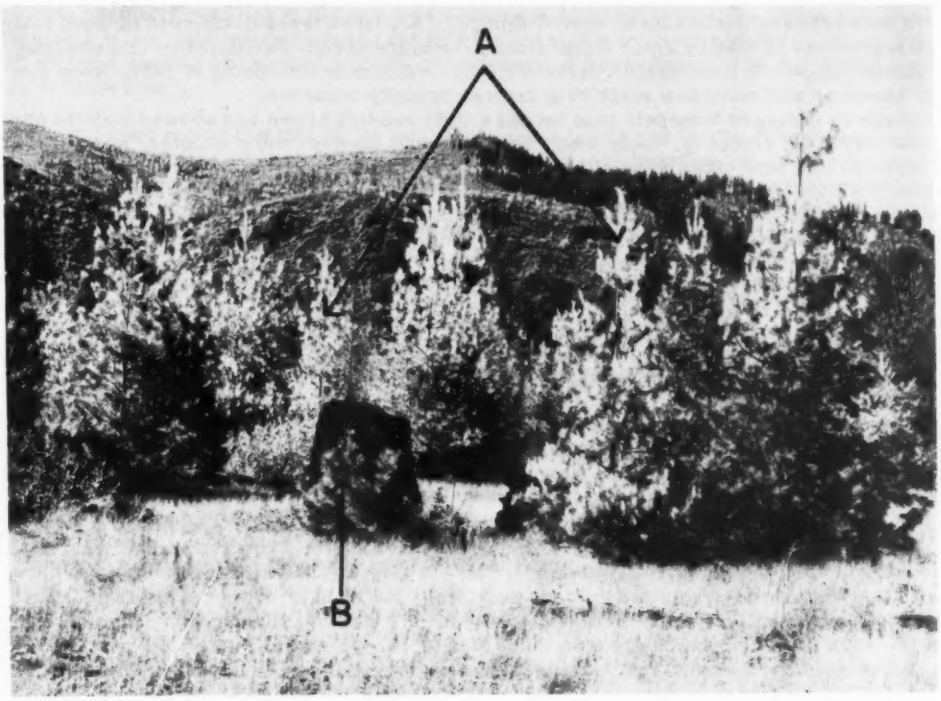

FIGURE 2. A group of planted white pine illustrating many degrees of drought injury from complete mortality (A) to no apparent injury (B).

FIGURE 3. This view of the plantation shows the severity of drought damage. No injury occurred on trees on the slope in the background.

Of the naturally regenerated species in the plantation, only grand fir, lodgepole pine, and western larch showed noticeable effects of drought. All larch trees discolored or shed their foliage regardless of size or age. Aside from premature needle discoloration and shedding, larch did not appear to be affected. However, observations in the spring of 1959, when new foliage appears, will show how much twig damage actually occurred.

Foliage on damaged lodgepole pine turned a light reddish brown and showed a slight shriveling but very little drooping. Only small seedlings and saplings were affected, and the majority of trees in this size class were killed outright. Larger trees apparently were not damaged.

Nearly all the grand fir in the plantation showed damage. On most trees the entire crown was affected. The needles turned a reddish brown and were slightly shriveled. On some trees the foliage in the upper crown and outermost portions of the branches had completely discolored, but normal appearing green needles were found interspersed with brown needles in the lower crown. Affected trees varied in height from 3 feet to approximately 25 feet.

The following conditions established drought as the cause of the plantation damage:

1. The plantation is located on a deep, gravelly, sandy soil except in a few places where sandy-loam soils predominated in the upper 1-foot horizon.

2. The area is nearly flat but has a slight west to southwest aspect.

3. No pathological agents or insects were associated with the damage. The roots even of trees killed to the ground line appeared quite healthy. Twigs and stem portions were dried out as a severed twig would dry when exposed to heat. Although no isolations were made, the wood showed no indications of cambial-killing fungi.

4. The 1958 growing season was abnormally hot and dry. Weather records at the permanent recording station about 5 miles to the north (Priest River Experimental Forest) showed that the monthly precipitation from October 1, 1957 to May 1, 1958 averaged somewhat above normal. However, total precipitation in May was only .69 inch, 1.49 inches below the mean. The average maximum temperature in May was 75.5° F, almost 10.5 degrees above the mean; and the average monthly minimum temperature was 41.1°, 4.5 degrees above the mean. June precipitation was above normal and fell chiefly before mid-June; but both maximum and minimum temperatures were also above normal. July precipitation was slightly below normal, and the average maximum and minimum temperatures were again above normal. Apparently August was the critical month. As usual for this time of the year, precipitation was limited -- only 0.9 inch, but the average maximum temperature was the highest on record. The average minimum temperature was also the highest except for 1915 and 1941, 27 and 0 years, respectively, prior to plantation establishment. For 18 days of this month maximum temperature went above 90° F.

5. Soil moisture appeared deficient early in the summer. Records have been maintained for the past 4 years at a permanent installation for recording soil moisture using fiber-glass units. The site has a west aspect and is located on the Priest River Experimental Forest at 4700 feet, almost 2500 feet higher than the plantation area. In previous years this recording unit showed that the soil moisture peak occurred in April, whereas in 1958 it was in February. The lowest readings were recorded in early September of previous years, but this year it occurred in mid-August and was lower than in any other recorded year. Determinations of available soil moisture were not made from field or laboratory tests for the plantation area. However, the top foot of soil was powdery dry in early September. The lower soil depths also appeared dry.

6. Streamflow also reflected drought. At the nearest recording gaging dam for streamflow (6 miles to the north at Benton Creek on the Priest River Experimental Forest) records have been maintained since 1939. Runoff started early in February. Though spring runoff continued into June, the discharge for June 1958 was one of the four lowest on records -- approximately one-half the mean for the month. Discharge during July, August, and September, which normally comes from ground water and very little from precipitation, was also one of the four lowest on record -- less than three-fourths of the mean for this period.

7. Hourly observations have been recorded at the Spokane Weather Bureau office on wind velocity, temperature, and relative humidity, along with other factors. From 1950 through 1954 the average number of hourly observations during July when winds exceeded a speed of 5 mph, temperatures were 80° F or greater, and the relative humidity was less than 30 percent, was 129.6. In July 1958 the number of observations with the same restrictions was 137. Of greater significance however, the number was 192 for August 1958, compared with an average of 95 for Augusts from 1950 through 1954. Though Spokane is about 65 air miles from the plantation, the Spokane records fairly represent the weather conditions prevailing throughout

the drought areas. These winds probably contributed abnormal moisture stresses. The root systems normally confined to the upper soil horizons in trees at this age had depleted moisture supplies. Water requirements could not be fulfilled either by the inadequate vertical root system or from insufficient moisture in the rhizosphere of this system. The inevitable result was drought injury.

Here is the reconstruction of the series of events leading up to the drought damage.

Snow left the ground early in March; above-normal precipitation continued until mid-April; and May was considerably hotter and drier than normal. Consequently growth started earlier than usual. Thus an early near maximum demand for soil moisture was made on a soil that has a very limited capacity for storing available moisture because of its gravelly sandy texture. Because of the site's location, there was little chance for soil moisture recharge from sources other than a high water table, which was not present. Neither was there an abundance of direct precipitation. Therefore available soil moisture was undoubtedly below normal by the end of May.

At high elevations soils started to dry out earlier than usual. At the low elevation plantation site, equivalent soil moisture stresses must have been reached at proportionately earlier dates. June precipitation was twice the normal, but most of it came during the first half of the month. Because precipitation alternated with above-average temperatures, it is questionable that much of it was effective in recharging the soil to meet subsequent demands.

Because the plantation is located on nearly flat ground with a west to southwest exposure, evapotranspiration normally would be high. June, July, and August temperatures were above the normal and contributed to abnormally high evapotranspiration stresses.

Soil moisture had apparently approached the critical level before August 1, judging from the early discoloration of larch in the area at that time. Winds came with the hotter-than-normal days in July and August.

Within the commercial white pine range drought damage was also observed in extreme northwestern Montana and northern Idaho; that is, on the Kootenai, Coeur d'Alene, St. Joe, and Kaniksu National Forests, but principally on the Kaniksu. Seedlings and saplings of both planted and wilding white pine showed symptoms of drought. Western larch and grand fir wildings were also affected. Damage was generally limited to a few individuals within each affected area. These areas, like the plantation, were deficient in soil moisture; they too had low available moisture storage capacities and limited soil moisture recharge potentials. Western larch everywhere was the indicator tree for drought, and wherever damage was most apparent, larch had defoliated earlier than normal.

Outside the range of white pine, drought injury was observed only in northeastern Washington from Colville east to the Idaho State line. Damage here was reported on lodgepole pine, Douglas-fir, and ponderosa pine seedlings and saplings. Considerable damage occurred to small lodgepole pine reproduction between Deer Park and Newport, Washington. Affected trees were growing on several sites, that were usually underlain by gravelly sandy soils with low water tables. No damage was observed in mature trees on any of the affected areas either within or outside the commercial range of white pine.

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DIE-BACK AND ROOT ROT DISEASE OF TAXUS SPP. IN INDIANA[1]

Lawrence R. Schreiber and Ralph J. Green, Jr.[2]

Abstract

A die-back and root rot disease of Taxus spp. in Indiana has been determined to be caused by Phytophthora cinnamomi Rands. Unfavorable environmental conditions, particularly high soil moisture, are necessary predisposing factors for infection and disease development.

INTRODUCTION

A serious die-back and root rot disease has caused severe losses in plantings of Taxus spp. in nurseries in Indiana for the past several years. The disease has occurred on cuttings in rooting benches and on rooted cuttings and older plants in lath houses and in the field. The symptoms include a gradual loss of normal green color and die-back of the aerial portion of the affected plants, with occasional wilting in young shoots. The root system shows extensive discoloration and a dry root rot. The tissues of the root and lower stem are streaked or discolored reddish-brown to black. Older plants seldom develop aerial symptoms until the root system is almost completely rotted.

This disease has been most severe in plantings on poorly drained sites or under conditions of high soil moisture. Growers frequently attribute losses to poor drainage, unfavorable weather, or poor quality stock. However, affected plants fail to recover from even the earliest symptoms when transplanted or when environmental conditions improve. Symptom development is progressive in older plantings and often follows natural drainage contours or develops from localized centers (Fig. 1). For these reasons it was assumed a pathogen was involved.

FIGURE 1. Phytophthora die-back and root rot of Taxus spp. in a lining out bed. Disease development is most severe in the poorly drained sections of the bed.

The symptoms observed correspond closely to those described by Crandall (2) and Crandall et al. (3) in the root rot and die-back of various hardwood and coniferous species caused by Phytophthora cinnamomi Rands. This disease is widespread in the Atlantic and southern States on nursery stocks, including Taxus cuspidata, T. baccata and T. media.

The results presented in this investigation include attempts to associate a specific pathogen with the disease symptoms observed in Taxus spp. in Indiana and a consideration of the influence of soil moisture upon disease development.

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[1] Journal paper No. 1435. Purdue University Agricultural Experiment Station, Lafayette, Indiana.

[2] Graduate Research Assistant and Associate Professor of Plant Pathology, respectively, Department of Botany and Plant Pathology, Purdue University, Lafayette, Indiana. The authors wish to acknowledge the assistance rendered by Dr. B. Zak, Southeastern Forest Experiment Station, Athens, Georgia, for the culture of Phytophthora cinnamomi Rands and Dr. A. T. Leiser, Department of Horticulture, Purdue University, for Taxus cuspidata plants for experimentation.

METHODS AND MATERIALS

Isolations were attempted from Taxus spp. plants showing early root rot symptoms. Tissue samples were taken from the roots and stem using one of the following isolation techniques:

1. Root and stem tissues were washed thoroughly with tap water, alcohol flamed, and plated in acidified (pH 4.5 to 5.0) and non-acidified potato-dextrose, corn meal, and oatmeal agars and incubated at 8°, 16°, and 24°C.
2. Root tissues were passed through several changes of sterile distilled water for 2 to 3 days prior to plating, as described by Crandall et al. (3).
3. Diseased root and stem tissues were minced and packed into holes bored into apple fruit and sealed. Isolations were made from the apple tissues 7 to 10 days later (1).

One- and 2-year-old rooted cuttings of Taxus cuspidata were inoculated with the organisms isolated from diseased plants. Inoculations were made either by 1) pruning roots to 3 inches and dipping the root system into an inoculum suspension, or 2) by placing mycelium and spores in wounds made in the lower stem. The potted plants were buried in soil in a cold frame and heavily watered. In a later experiment, rooted cuttings were inoculated by working inoculum into the soil around the roots. Inoculations were made with the organism consistently isolated from diseased Taxus spp. plants and tentatively identified as Phytophthora cinnamomi, and with a known isolate of P. cinnamomi for comparison. The inoculated plants were held at 70°F in controlled climate facilities. After 7 weeks, replications from each treatment were flooded for 7 days and 28 days, respectively. Two weeks after flooding the plants were observed for symptom development. Check plants consisted of non-inoculated, flooded and non-flooded treatments.

Microscopic and cultural comparisons were made between the isolate from diseased Taxus spp. plants and a known isolate of P. cinnamomi grown on potato-dextrose and corn meal agars and sterile wheat grains. Attempts were made to induce sporangia and zoospore formation by washing mycelium removed from sterile wheat grains repeatedly with sterile distilled water and "starving" the cultures in water.

RESULTS

An organism tentatively identified as Phytophthora cinnamomi was consistently isolated from the root and stem tissues of diseased Taxus spp. plants, utilizing the apple fruit technique. The other isolation methods yielded various fungi and bacteria, including Fusarium sp., but none proved pathogenic to Taxus cuspidata plants.

The results of studies of the effect of soil moisture level on disease incidence under controlled conditions are presented in Table 1.

Table 1. The effect of flooding on the development of die-back and root rot of Taxus spp.

Treatment	Non-flooded	Flooded	
	7 weeks	7 days	28 days
Inoculated			
Isolate-Taxus sp.	0	+[a]	+
P. cinnamomi	0	+	+
Non-inoculated	0	0	0

[a] + - chlorosis, wilting, and needle drop
0 - no symptom development

No disease symptoms appeared in any treatment within 7 weeks under controlled temperature conditions and normal watering. Characteristic symptoms of the die-back and root rot disease developed within 2 weeks after inoculated pots were drained in both the 7 day and 28 day flood treatments (Fig. 2). Inoculated plants in the flood treatments exhibited extensive root rot and failed to develop additional adventitious roots. Inoculated plants that were not flooded

showed some root rot but recovery was evident by the development of new roots from the root collar.

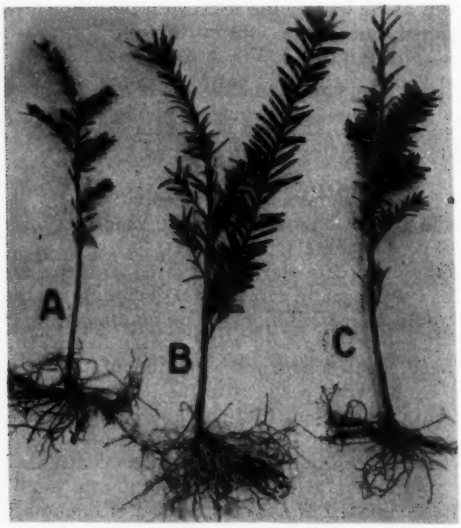

FIGURE 2. Root rot symptoms on 1-year-old Taxus cuspidata plants after inoculation and flooding. A-- Inoculation with P. cinnamomi from short leaf pine. B -- Non-inoculated control. C -- Inoculated with P. cinnamomi from Taxus sp.

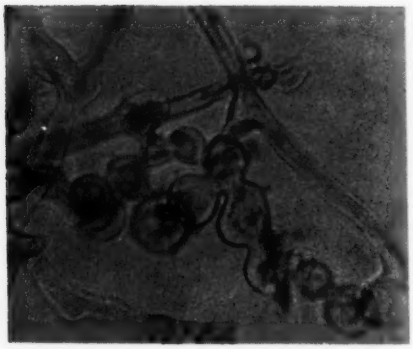

FIGURE 3. Hyphae of P. cinnamomi showing lateral protrusions and terminal swellings.

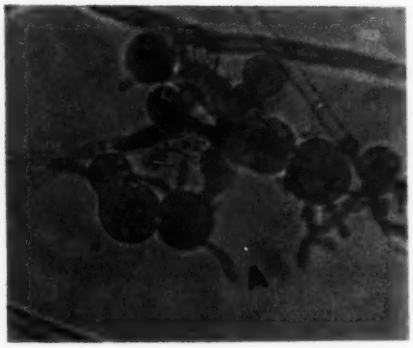

FIGURE 4. Chlamydospores of P. cinnamomi grown on sterile wheat grains. A -- Germ tube from germinating spore.

The cultural characteristics of the isolate from diseased Taxus spp. plants are as follows: The hyphae are hyaline, 3.2 to 8 μ in diameter, and non-septate in young cultures, with increasing septation as the culture ages. Irregular hyphal swellings or protrusions occur laterally and on hyphal terminals (Fig. 3). Cultures grown on sterile wheat grains and then "starved" in sterile distilled water readily form chlamydospores singly or in clusters on short stalks. These spores are spherical, thin walled, and range from 6.0 to 28.8 μ in diameter. The chlamydospores germinate with one to three germ tubes (Fig. 4).

Sexual structures and sporangia were not observed in any of the cultures studied. However, Leonian (4) states that P. cinnamomi is readily recognized by its typical hyphal swellings, by the formation of chlamydospores, and by the lack of sporangia on solid media.

CONCLUSIONS

The die-back and root rot disease of Taxus spp. observed in nursery plantings in Indiana is caused by a pathogen identified as Phytophthora cinnamomi Rands. It is apparent that infection and disease development is dependent upon unfavorable soil moisture or other environmental conditions detrimental to root development and plant growth.

Phytophthora cinnamomi has not previously been reported on Taxus spp. in Indiana or other States in the Midwest. Growers have not been concerned with sources of planting stock. As a consequence, the causal organism has undoubtedly been introduced into many commercial plantings on rooted cuttings and liners, in infested soil, and by other means.

The results of this investigation emphasize the need for careful consideration by nurserymen of the source of planting stock and of propagation practices used to minimize the losses from the die-back and root rot disease.

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SEVERITY OF TOMATO SOIL-ROT CAUSED BY RHIZOCTONIA SOLANI AS INFLUENCED BY FORMER PASTURE CROPS

Norman C. Hayslip and Robert E. Stall[1]

Summary

Soil rot of tomato fruit caused by Rhizoctonia solani Kuehn was much less severe where Pensacola bahiagrass and pangolagrass alone preceded the tomato crop than where these grasses plus white Dutch clover preceded the tomatoes. These results were obtained in each of three experiments on Immokalee fine sand in the Indian River area of Florida. Yields of graded tomatoes were higher in the grass alone plots in two of the experiments.

Most tomatoes raised for mature green harvest on the sandy soils of south Florida are produced on virgin soil. Experience has taught farmers that problems increase sharply when tomato crops are repeated on the same land. One of the most serious limitations in the production of tomatoes on "old land" has been an increase in soil rot of fruit caused by Rhizoctonia solani Kuehn. This soil-borne pathogen invades fruit which are in contact with the soil. During wet seasons with warm temperatures soil rot can almost totally destroy the crop (Figure 1). As a result of this and other problems "old land" has been allowed to revert to natural vegetation, or is planted to improved pastures.

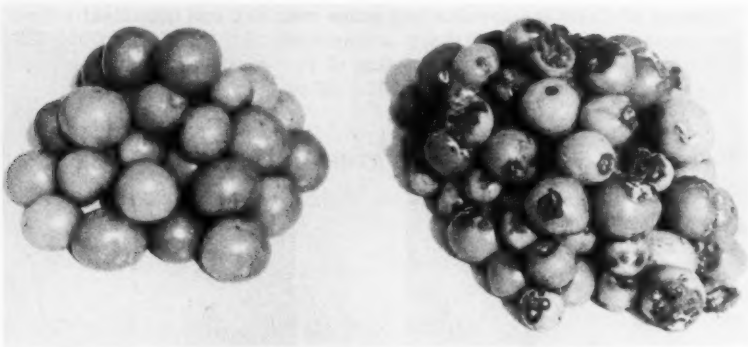

FIGURE 1. Tomato soil rot can be severe in areas where clover precedes the tomato crop. Tomatoes shown above were harvested from plot that had been in pangolagrass plus white Dutch clover. More than half of the fruit was rotted.

Since the quantity of virgin soil suitable for tomato production is rapidly becoming depleted, farmers soon will be forced to return to some of the previously used areas. In an effort to find a way to produce tomatoes on the same soil at reasonable intervals, a tomato-improved pasture rotation study was begun in 1950. Several phases of this rotation study have been reported previously (1, 2). The results reported in this paper indicate that the type of improved pasture used influences the amount of soil rot developing on the succeeding tomato crop.

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MATERIALS AND METHODS

Three separate experiments are reported. In all trials the soil type was Immokalee fine sand. There had been at least one tomato crop raised on this land before grass and grass plus white clover were established. The grass plots were allowed to produce a heavy cover throughout the period, whereas the grass plus white clover plots were mowed and burned each fall to remove the heavy grass growth. Removal of the grass was necessary to promote good clover growth during the winter and spring months. As the clover growth decreased in the spring, the grass grew rapidly and produced a heavy cover during the summer months. After allowing the improved pastures to grow on the plots for a suitable period, the plots were prepared for a tomato crop. Preparation for tomatoes began about 2 months before transplanting the tomato seedlings. The forage was removed by mowing and burning. The plots were then disked several times. The areas were limed and treated for cutworm, mole cricket and wireworm control before planting tomatoes. If the soil was dry at planting time, it was moistened by overhead irrigation. Standard fertilization, cultivation and spraying methods were used in all experiments. Fruits with soil rot and yields were recorded at harvest.

EXPERIMENT 1 -- FALL, 1956

The two types of improved pastures in this experiment were Pensacola bahiagrass alone and Pensacola bahiagrass plus white Dutch clover. Plots measuring 50 feet by 50 feet were replicated three times. After 2 years in these improved pastures, the tomato variety Homestead-2 was transplanted September 13, 1956, and fruit harvested December 7 and 18. Prolonged heavy rainfall (14.28 inches) occurred frequently during the growth of this tomato crop.

Table 1. Percent of tomato fruits affected by Rhizoctonia solani when preceded by Pensacola bahiagrass and Pensacola bahiagrass plus white clover. Fall, 1956.

	Replication			
	A	B	C	Ave.
Clover + Grass	26	32	16	25
Grass alone	2	3	1	2

Counts of tomatoes with soil rot were obtained from a random sample of 200 fruits per plot during the second harvest. The percent of fruit affected is listed in Table 1. The ungraded yield of tomatoes was 35 percent larger in the grass plus clover plots, but the amount of soil rot was much more severe than in the bahiagrass alone plots.

EXPERIMENT 2 -- SPRING, 1958

In this experiment virgin soil was cropped to tomatoes in the spring of 1954. Following this crop, pangolagrass was planted June 9, 1954, and white clover seeded in alternate strips November 10, 1954. There were two 50 by 193-foot plots of pangolagrass alone and two of pangolagrass plus white clover. These plots were maintained in these pastures for 3 years. One-half of each plot was prepared for spring tomatoes in the winter of 1957-58. Twelve tomato varieties were transplanted February 27, 1958 in each plot. Soil rot levels were determined by

Table 2. Percent of tomato fruits affected by Rhizoctonia solani when preceded by pangolagrass and pangolagrass plus white clover. Spring, 1958.

		Replication												
	Rep.	1	2	3	4	5	6	7	8	9	10	11	12	Ave.
Clover + Grass	A	30	51	38	30	37	35	27	33	41	41	40	40	37
Grass alone	A	7	10	13	18	11	18	14	19	19	30	10	14	15
Clover + Grass	B	33	48	42	43	31	40	40	51	41	43	35	43	41
Grass alone	B	16	33	18	12	7	23	10	22	19	24	10	17	18

counting diseased fruits from each variety during each of three harvests. Each tomato variety was considered a sample, making twelve samples in each main plot. Rainfall (12.22 inches) was heavy during this crop. Data are listed in Table 2.

It should be emphasized that in every sample there was less soil rot on plots which previously had been maintained in grass alone. Twenty-one percent more graded fruit were produced in the grass alone plots. There were no differences in ungraded yields.

EXPERIMENT 3 -- FALL, 1958

The north half of each of the plots described in Experiment 2 was prepared for tomatoes in the late summer of 1958. This was 3 1/2 years after the pastures were established. As in Experiment 2, twelve tomato varieties were raised in each plot and used as separate samples for soil rot counts. There was less soil rot and less rainfall (9.29 inches) in this experiment than in the previous two experiments. Data obtained are listed in Table 3.

Table 3. Percent of tomato fruits affected by Rhizoctonia solani when preceded by pangolagrass and pangolagrass plus white clover. Fall, 1958.

	Rep.	Replication 1	2	3	4	5	6	7	8	9	10	11	12	Ave.
Clover + Grass	A	3	22	13	15	11	28	22	12	15	16	18	13	16
Grass alone	A	1	7	3	2	2	6	2	0	7	6	3	2	3
Clover + Grass	B	18	33	20	15	18	33	22	15	18	9	24	12	20
Grass alone	B	2	7	1	6	1	7	0	3	4	1	4	4	3

Again, every sample had much less soil rot in the grass plots than in the grass plus clover plots. Also, the yield of graded fruit was 19 percent higher in the grass plots. There were no differences in ungraded yields.

DISCUSSION

This work further emphasizes the importance of the effect of cropping sequences on the population of soil-borne fungi. All three experiments were started on virgin soil in which the population of Rhizoctonia solani is inherently low. Under one sequence, tomatoes rotated with white clover plus grass, the population of the pathogen rose to a disastrous level. Under another sequence, tomatoes rotated with the grass alone, the population remained at a low level.

It appears that white clover was responsible for the high level of the pathogen. The clover could have caused a "build-up" of the organism or it could have maintained the high level caused by the tomato crop in the rotation. At any rate, farmers are advised against planting un-staked tomatoes on the sandy soils of south Florida where clovers are growing. Since R. solani attacks most other vegetables, the data presented here may be interpreted to mean that an increased problem with the organism may occur on other vegetables planted after white clover.

On the other hand, a tomato and improved pasture-grass rotation is encouraging. The amount of soil rot in such a rotation was below economic significance in two of the three experiments. Further studies are planned to determine the effect of various rotations on the level of the pathogen. These rotations will be started on virgin soil.

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FOLIAGE INFECTION OF TOMATO AND EGGPLANT BY VERTICILLIUM[1]

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Abstract

Foliage inoculation experiments in the greenhouse with tomato and eggplant showed that Verticillium albo-atrum Reinke & Berth. produced hydathodal infections of the leaves which resulted in some instances in a systemic invasion of the plant. Leaf lesions were at first marginal and chlorotic, but enlarged and became progressively necrotic, resulting in premature leaf abscission. The reaction of resistant varieties to foliage infection was similar to that resulting from root infection. The optimum temperature for foliage infection was essentially the same as that for root infection.

INTRODUCTION

In 1945, an unknown defoliation disease of tomato developed in the fungicide test plots at Geneva, New York. The fungicides reduced the defoliation, some to a greater degree than others. The disease was not identified at the time, but the foliage symptoms resembled those of Verticillium wilt. Verticillium albo-atrum Reinke & Berth. was isolated from similar lesions on leaves taken from tomato plants growing in a field at Medina, New York in 1950. Attempts to recover the fungus from the roots and stems of these plants failed, suggesting that aerial parts of the plants, particularly the foliage, might be sites of primary infection and that the root system or injured portions of the crowns of some plants are not the only infection sites. Consequently, experiments were designed to determine whether V. albo-atrum could incite local lesions on leaves of tomato and eggplant. In addition, it was deemed advisable to determine if such local infections of the foliage could induce a systemic invasion of the plant. The influences of air temperature and age of plants on infection were also investigated.

MATERIALS AND METHODS

One isolate of V. albo-atrum, obtained from the roots of a diseased tomato plant collected at Medina, New York and proved to be pathogenic via root inoculation, was used in all of the experiments.

All tests plants of tomato and eggplant were derived from hot-water treated seed that was planted in sterilized quartz sand and the plants pricked therefrom into sterilized pots containing pasteurized soil. Susceptible tomato varieties used in these studies were Gem, Fireball, Red Jacket, Manalucie and Blackhawk, the latter two being resistant to Fusarium wilt. Verticillium-resistant varieties included Loran Blood, Moscow VR and Geneva 11. Black Beauty served as the eggplant suscept.

Maximum spore production was obtained by growing the fungus in a liquid medium contained in a Roux culture flask. The medium consisted of the following ingredients, in grams per liter of distilled water: KNO_3 -- 3.00, KH_2PO_4 -- 1.00, KCl -- 0.50, $MgSO_4 . 7H_2O$ -- 0.50, ferric ammonium citrate -- 0.01, glucose -- 15.00, sucrose -- 15.00. Each culture flask containing 150 ml of the above medium was plugged and autoclaved for 25 minutes at 250° F. After cooling, the flasks were seeded with mycelium cut from a potato-dextrose agar culture of the fungus. Each flask was shaken several times each day for 3 days to break up and distribute the mycelial fragments throughout the medium. Thereafter, the flasks were stacked on their flat sides and allowed to remain undisturbed for 8 to 10 days at room temperature. Almost all of the mycelium grew on the surface of the liquid medium, producing a multitude of spores which settled to the bottom of the flask. The importance of surface area of liquid in spore production was indicated by the fact that the same volume of liquid in a 500 ml Erlenmeyer flask, inoculated and treated the same as that in the Roux flasks, did not yield nearly as many spores. The surface area of the liquid nutrient in the Roux flask, however, was roughly 2 1/2 times that in the Erlenmeyer flasks. Culturing the fungus in the same volume of liquid nutrient contained in Erlen-

[1] Approved by Director of the New York State Agricultural Experiment Station, Geneva, New York, as Journal Paper No. 1164.

meyer flasks that were mechanically shaken produced the least spores.

Practically pure suspensions of spores were obtained by filtering the suspended contents of each flask through several layers of cheesecloth. Traces of nutrients and toxic by-products were removed by washing with distilled water in three centrifugations. The washed spores were resuspended in distilled water. The concentration was adjusted, with a haemocytometer, to approximately 2.0×10^6 spores per ml of spore suspension for each inoculation. Spore germinations were recorded by counts obtained from culture slides containing droplets of the suspension kept in the inoculation chamber.

Each plant was individually inoculated. Foliage was sprayed with the spore suspension, using either a deVilbiss atomizer or a paint spray gun, depending upon the size and number of plants in the experiment. Leaves were sprayed until droplets formed. In the initial experiments no attempts were made to exclude soil contamination by the spores atomized on the leaves. In some of the succeeding experiments aluminum foil was used to cover the top of the pot, but there were reasons to suspect that this did not entirely prevent soil contamination. The best insurance against soil contamination was obtained by placing the pots in polyethylene bags which were tied rather tightly around the stems and allowed to remain. After the pots were removed from the moist chamber, the bottoms of the bags were cut so that they fitfed over the saucers in which the pots were placed to be subirrigated.

Evidence for and locations of infection were based upon fungus isolations and histological examinations of diseased plant parts. Plant structures for fungus isolation were washed with tap water and then surface disinfected with commercial sodium hypochlorite (Clorox) for 5 minutes at dilutions of 1 to 9 for leaves and 1 to 8 for roots and stems. Evidence that the spores of the original suspension which did not penetrate tissue either died or were killed by the surface disinfectant is illustrated in platings wherein the fungus was re-isolated only from leaf areas that exhibited symptoms (Fig. 1). Systemic invasion of a plant was ascertained by dia-

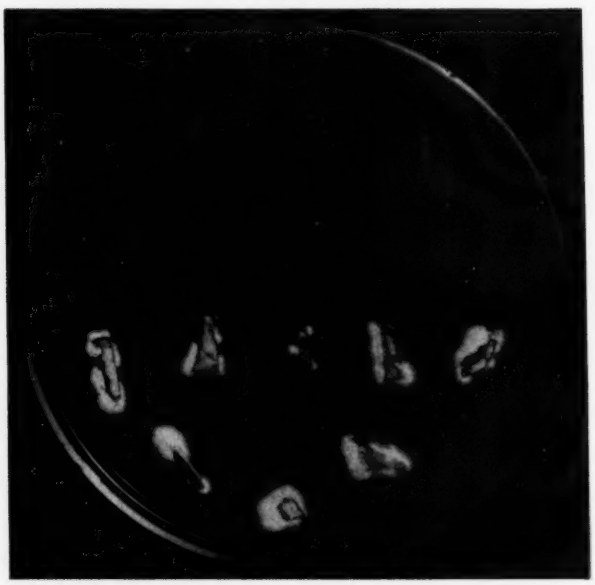

FIGURE 1. Platings of tissue sections from the same leaflet previously atomized with a spore suspension of V. albo-atrum to show the efficacy of surface disinfection. Sections in the upper half of the plate from symptomless areas and those in the lower half from symptomatic areas. One tissue section contaminated.

gramming the plant with respect to the positions of the tissues isolated from, and then plotting the positive isolations. Material for histological examination was fixed in FAA, sectioned at 10 to 15 microns and stained by Stoughton's thionin-orange G method.

RESULTS

Symptoms of Foliar Infection

Foliar inoculations confirmed the suspicion, based on field observations and fungus isolations, that V. albo-atrum could establish itself in foliage of tomato and eggplant. On susceptible varieties, the primary symptom is a chlorotic area along the margin of the blade, suggesting entrance through hydathodes (Fig. 2). Chlorotic areas were observed away from the mar-

FIGURE 2. Numerous chlorotic lesions on leaves of susceptible Gem variety of tomato 25 days after foliage inoculation with V. albo-atrum. Non-inoculated leaf on left.

gin of the blade, rarely in tomato and only occasionally in eggplant, suggesting penetration through some avenue other than hydathodes, possibly through wounds. Infection progressed along a lateral vein toward the midrib of the leaf or leaflet, usually resulting in a V-shaped lesion of chlorotic tissue that became progressively necrotic. Eventually the entire leaf dried, and severely infected leaves abscised prematurely.

Symptoms also appeared on the foliage of resistant tomato varieties, but they were reduced in intensity. Such lesions were marginal and chlorotic but not very numerous. The time required for symptoms to appear was at least twice as long as with susceptible varieties (Figs. 2 and 3). Lesions progressed very slowly, and in most instances involved only a limited area. Leaf abscission seldom occurred.

Penetration of Foliage and Subsequent Systemic Fungal Invasion

The almost complete restriction of leaf lesions to the margins suggested that infection might have occurred via hydathodes. This possibility was supported by the observed progressive movement of the symptoms inward from the margins, seemingly along veins. Histological evidence obtained from stained sections confirmed this supposition (Figs. 4 and 5). The fungus was found sparingly in the protoxylem of the leaf veins that terminated in the epithem. Plugged vessels, however, were observed in great abundance, especially at the distal end. Furthermore, histological evidences of the fungus and vessel plugging diminished with the distance from the leaf margins. No fungal hyphae were observed in any but tracheal tissues of leaves with primary symptoms. Heavy sporulation has been observed on abscised leaves with advanced symptoms when they were kept in a moist atmosphere. This would indicate hyphal growth in other tissues, as has been pointed out previously by other workers.

Attempts to infect cotyledonary leaves by spore atomizations failed. This precluded the use of such an inoculation as a quick method of evaluating new germplasm and breeding lines for re-

FIGURE 3. Sparse development of chlorotic lesions on leaves of resistant Loran Blood variety of tomato 62 days after foliage inoculation with V. albo-atrum. Non-inoculated leaf on left.

FIGURE 4. Hypha in annular trachea of vein in leaflet of Gem tomato infected via the hydathode.

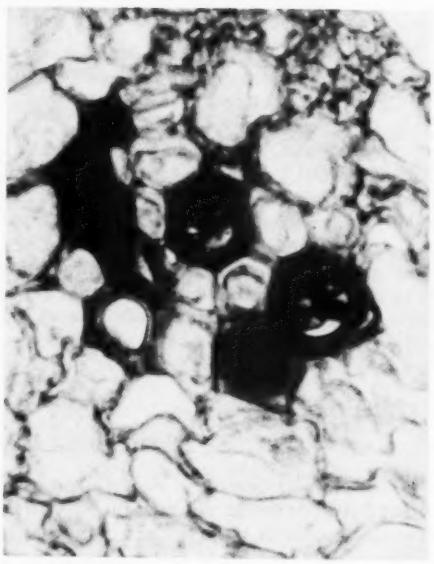

FIGURE 5. Transection of leaflet vein of Gem tomato infected via hydathodes, showing the plugged vessels.

sistance. Guttations were observed in cotyledonary leaves of the susceptible varieties of tomato, which indicated the presence of hydathodes. The reason for the infection failures was not determined.

The possibility of systemic invasion, suggested by the nature of the foliage syndrome and strengthened by the histological evidence, became real upon investigation. In one experiment

involving three susceptible varieties -- each with four plants in four different age groups -- systemic invasion resulting from foliage inoculation occurred in 9 of the 48 plants. Isolations attempted from various positions on the systemically invaded plants were positive from the roots as well as from the stems in five of the plants. This indicated soil contamination or an unlikely rapid downward growth of the fungus to the roots. In four of the plants, however, the fungus was isolated from the stems and leaves of the post-inoculation growth but not from the roots. In the 39 non-systemically infected plants, the pathogen was isolated only from the leaves present at the time of inoculation.

Systemic invasion was confirmed in another experiment in which greater precautions were taken to avoid soil contamination by enclosing the entire pot in a polyethylene bag, as described under methods. Sixteen plants of each of two susceptible and two resistant varieties at the 7 to 9 true-leaf stage were used. Eight plants of each variety were inoculated by atomizing the spore suspension on the fourth and fifth leaves, taking care not to contaminate the other leaves and preventing subsequent contamination by enclosing each inoculated leaf in a small polyethylene bag. On the remaining eight plants of each variety, the spore suspension was applied to the entire foliage. Four of the eight plants in each inoculation group were held for 48 hours in a moist chamber at approximately 70°F. The other four plants were held for the same period of time at 80°. All inoculated susceptible plants developed local lesions along the leaf margins. Six of the 16 plants at 70° showed unmistakable evidence of systemic invasion; whereas, only two of the 16 plants held at 80° did. Only one-fourth of the systemically invaded plants belonged to the group in which only the fourth and fifth leaves were inoculated. The inability to recover the fungus from the roots and hypocotyls of these plants precluded any possibility of systemic invasion via the root system. No systemic invasion was noted in resistant varieties treated in the above manner and, as pointed out in previous experiments, local lesion development was sparse and delayed in appearance.

Temperature in Relation to Foliar Infection

The amount of infection was influenced by the temperature of the air during the 48-hour period that plants were held in a saturated atmosphere following inoculation. Plants held at 66° to 68° F for 48 hours and then incubated at 80° developed an average of 20 lesions per leaf; whereas, plants held at 78° to 80° and similarly incubated developed only 12 lesions per leaf. Although spore germination proceeded at a faster rate at the higher temperature, at the end of 24 hours both reached the maximum of 92 percent. Fifty percent more leaf abscission occurred in the susceptible varieties at the lower temperature, probably as a result of the increased infection.

Leaf Maturity in Relation to Infection

The length of the incubation period and the frequency of lesions appeared to be related to the maturity of the leaf. This was observed in experiments in which seeds were planted at scheduled intervals to obtain plants of different ages which, consequently, would have certain leaves in common that would be of different maturities; namely, the first, second and third leaves. Symptoms appeared on such leaves of plants in four age groups possessing 2 to 3, 4 to 6, 10 to 12 and 12 to 14 fully expanded leaves at the time of inoculation of 10, 10, 15, and 20 days, respectively. Lesions were less numerous on the leaves of the oldest plants. It should be pointed out, however, that the few lesions were found on the very youngest leaves, those that were not completely expanded at the time of inoculation. These results were confirmed by repeated experiments.

DISCUSSION

Foliage infection of tomato and eggplant by spores via the hydathodes shows similarities to the root infection phase of the disease and may clarify some controversial points. Although foliage infection apparently begins as a local lesion, the incitant actually invades the vascular system of the leaf, apparently against the flow of the sap stream, and may ultimately produce a systemic disease. The low incidence of systemic invasion may be largely the result of premature abscission of the leaf, thereby preventing the organism from reaching the stem. The optimum temperature of 70°F for foliage infection is similar to that for root infection. The similarity in reaction of resistant varieties to foliage and root infections; namely, long incubation period and limited advance of the symptoms, indicates that the resistance factor may also reside in the foliage, not solely in the roots as suggested by Keyworth (3). Spores produced on senescent or dead Verticillium-infected plant tissues, as observed recently by Isaac (2) and oth-

ers (1, 4, 5, 6), could be dispersed by air and other agencies to susceptible foliage of plants growing in clean soil and thus establish soil infestations in remote areas much faster and to a greater degree than if the spores settled on bare soil.

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EFFECTS OF DISEASES ON CASTORBEANS IN MISSISSIPPI

William J. Stone and Thomas W. Culp[1]

High yields of castorbeans (Ricinus communis) in 1956 and previous years indicated that this crop has the potential of providing Mississippi farmers with another source of income. However, yield losses from diseases were extremely heavy during 1957 and 1958. Apparently the diseases were made worse by the high rainfall and humidity. Diseases considered to be the most severe were Alternaria capsule mold, Alternaria ricini (Yoshii) Hansford; gray mold, Botryotinia ricini (Godfrey) Whet.; and bacterial leaf spot, Xanthomonas ricinicola (Elliott) Dowson. Cercospora leaf spot, Cercospora ricinella Sacc. & Berl., and Alternaria leaf spot (Alternaria ricini) were observed, but caused only minor damage. A wilt seemingly caused by a bacterium did considerable damage at Poplarville and State College, Mississippi.

Alternaria capsule mold and leaf spot: Alternaria capsule mold was the limiting factor in castorbean production in Mississippi in the past 2 years. Alternaria ricini may attack capsules as they approach maturity and completely prevent seed development or, if seed have developed, it may weaken or kill the pedicel of the capsule and cause the seed to drop. Its attack may result in almost complete loss in yield of the more susceptible varieties. The severity of the disease is illustrated in Figure 1. Resistance to Alternaria capsule mold has not been found in any of the breeding material grown at Stoneville. The variety Baker 296 and one ornamental strain, MW-1, possessed the greatest tolerance, but about 25 percent of the seed of even this variety and strain were lost. The severity of Alternaria capsule mold is reflected in the yields obtained. Baker 296 produced about 1000 pounds of seed per acre in 1958. Yields of other tested varieties were much lower; several produced less than 100 pounds of seed per acre. In Table 1 the 1957 and 1958 yields of six castorbean varieties are compared with the 1956 yields when Alternaria capsule mold was not a problem. It was estimated from these data that the disease caused yield reductions of about 70 to 85 percent in 1957 and 1958, respectively.

Table 1. Mean yield in pounds per acre of seven castorbean hybrids and varieties grown at Stoneville, Mississippi, from 1956 through 1958[a].

Variety or hybrid	1956	1957	1958
415 Hybrid	3693	1015	446
Pacific Hybrid 6	2995	1286	381
Cimarron Hybrid	2930	914	558
Cimarron	2469	1037	699
US 65	3148	598	99
Dawn	2292	632	245
Baker 296	--[b]	1425[b]	1043[b]
Mean	2921	914	405

[a]Each figure is the mean of 4 replications.
[b]Not grown in 1956 yield tests. Not included in averages.

Alternaria leaf disease has been of minor importance in Mississippi. This was surprising, since leaf spot was reported to cause relatively severe epiphytotics in other areas with comparatively high relative humidity. Alternaria was found attacking cotyledons of castorbean seedlings in 1957, but not in 1958. Mature leaves of plants have had minor damage, with Alternaria appearing to move in following initial infection with bacterial leaf spot.

Gray mold: Gray mold is one of the more severe diseases of castorbeans during seasons of abundant rainfall and humidity. The disease is a typical Botrytis blight on the racemes.

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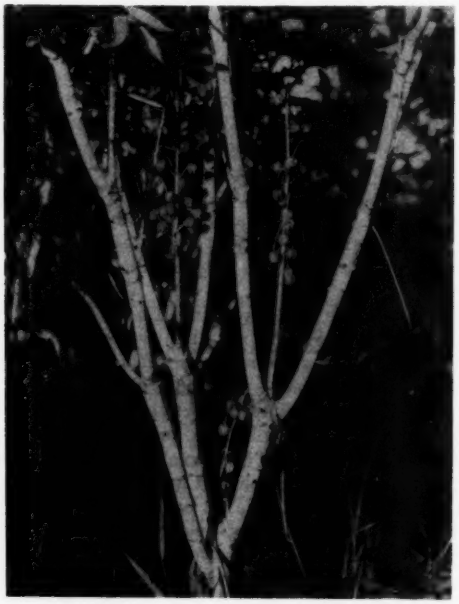

FIGURE 1. Castorbean racemes infected by Alternaria ricini showing extent of capsule drop it caused.

FIGURE 2. Castorbean raceme infected by gray mold.

FIGURE 3. Castorbean plants attacked by wilt at Poplarville, Mississippi.

When weather conditions are optimum for its development the fungus attacks the racemes at all stages of development; growth of the mold is heavy and superficial and infected racemes are almost completely destroyed. An infected raceme collected from a commercial planting is illustrated in Figure 2. The leaves and stems have also been reported to be attacked by the gray mold organism (1, 2).

The most severe outbreak of the disease in Mississippi occurred in 1958 in a commercial field of approximately 100 acres near Cleveland. An estimated 25 percent or more of the racemes in the planting were infected by gray mold. This same field had been planted to castorbeans during two previous seasons, and the disease had increased each year. It appeared that the continuous cropping with castorbeans had caused a tremendous increase of the pathogen.

Occurrence of gray mold in other areas of the State has been comparatively light. However, there was some infection wherever castorbeans were grown.

Control of gray mold is difficult. No resistance or tolerance has been found in the present breeding material. Because of the difficulty of obtaining coverage of the large dense plants with an acceptable fungicide, it has not been feasible to use chemical control. Rotation should be helpful in controlling the disease.

Bacterial leaf spot: Epiphytotics of bacterial leaf spot often develop but do not appear to cause severe yield losses in Mississippi. Numerous lines of castorbeans possessing good resistance are now available; thus it should be possible to control the disease by the development of resistant varieties.

Cercospora leaf spot: Cercospora leaf spot has been a minor problem. Often it causes greatest damage during a program of screening for resistance to other leaf diseases, because of the difficulty in evaluating the severity of attack of the disease under study.

Castorbean wilt: In 1958 a severe wilt of castorbeans was observed at Poplarville (Fig. 3) and later at State College, Mississippi. Symptoms of the disease agreed very closely with a description of a brown rot, or wilt, of castorbeans reported by Smith and Godfrey (3) as caused by Xanthomonas solanacearum (E. F. Smith) Dowson. This organism has been reported as having a wide host range.

Diseases have been found the limiting factor in castorbean production in the Mississippi area. Since it is unlikely that chemical controls can be used economically, disease-resistant varieties must be found or developed if castorbeans are ever to become a commercial crop in Mississippi.

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SURVEY OF IRRIGATION WATER FOR THE PRESENCE OF PHYTOPHTHORA SPP. PATHOGENIC TO CITRUS

L. J. Klotz, Po-Ping Wong, and T. A. DeWolfe[1]

Abstract

Twelve canals and three reservoirs which supply irrigation water to citrus groves were tested for the presence of Phytophthora spp. that are pathogenic to citrus. After 1 year of continual sampling with a lemon-trap method it was found that all 12 canals and one reservoir harbored one or more species of Phytophthora. The species prevalent during the year varied with the seasonal changes in the temperature of the water.

INTRODUCTION

Phytophthora citrophthora (R. E. Sm. & E. H. Sm.) Leonian, P. parasitica Dast., P. syringae Kleb., P. hibernalis Carne, and P. megasperma Drechs. have been found to be pathogenic either to the above or below ground portion, or both, of commercially grown citrus trees in California (1, 7). Some are found to be widespread in soil of major citrus producing areas of the world. The purposes of this water-testing work, initiated in the fall of 1957, were to determine the prevalence of Phytophthora spp. pathogenic to citrus in irrigation water of open canals and reservoirs in five southern California counties, namely--Riverside, San Barnardino, Los Angeles, San Diego, and Orange; and to investigate the possibility that Phytophthora-infested citrus groves introduce the fungus into the open canals by run-off water, either as excess irrigation water or as rain.

MATERIALS AND METHODS

The method of testing for the presence of Phytophthora spp. consisted of placing 5 to 20 lemons (silver stage) in a perforated plastic bag and suspending them in the canal or reservoir for 2 weeks. If the fungi were present, their zoospores infected the fruit and caused a typical brown rot. The bags of fruits were then collected, brought to the laboratory, washed, and those with brown rot lesions were cultured. Depending on the length of the canal, the number of traps placed at intervals in the canal varied from two to five. Where possible a trap was also placed at or near the source of the canal water. Usually each location was sampled once every 2 weeks.

Boiled hempseeds were added in each trap to increase the probability of trapping any Phytophthora spp. (9), and oranges for P. hibernalis (5). With this method lemons were found still to be the most satisfactory trapping agent for Phytophthora citrophthora, P. parasitica and P. syringae in water.

RESULTS

Of the 12 canals tested during the period of September 1957 to September 1958, all yielded Phytophthora spp. at one time or another, some more consistently than others. The most commonly distributed and frequently recovered species was P. citrophthora (11 canals), less frequently, P. parasitica (5 canals); and least frequently, P. syringae (2 canals). P. hibernalis and P. megasperma were not found.

Some canals yielded more than one species of Phytophthora. The following is a grouping of the number of canals in relation to the species of Phytophthora isolated:

Four canals yielded P. citrophthora only.
Four canals yielded both P. citrophthora and P. parasitica.
Two canals yielded both P. citrophthora and P. syringae.
One canal yielded P. citrophthora, P. parasitica, and P. syringae.
One canal yielded an unidentified Phytophthora sp. that tolerated an exceptionally high temperature range, being able to withstand 45°C incubation. Under greenhouse conditions it was capable

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of producing gummosis lesions both in the bark and on the cambium layer of the trunk of 1-year-old seedlings of Cleopatra mandarin, Standard sour orange, Jameson sweet orange, and Sampson tangelo.

Where more than one species of Phytophthora were found in the canal during the several samplings the species isolated seemed to be related to the temperature of the water prevailing at that time of the year. During winter and spring months, when the water temperature was between 6° and 17°C, P. syringae was isolated. During the warm summer and early fall months, when the water temperature ranged from 17° to 31°, P. parasitica was isolated. However, P. citrophthora was recovered all through the year, with the water temperature ranging from 6° to 31°. These findings coincide closely with the temperature range at which these fungi grow in artificial media (1).

In five canals where it was possible to set the lemon traps at the source of water supply, no Phytophthora spp. were recovered. However, as the canals passed through citrus areas where excess irrigation water or rain could drain into the canals, the fungi were readily isolated from the water thus contaminated. Soil samples collected and cultured (5) from four separate paths of run-off water that drained into irrigation canals also yielded the fungi. All the soil samples yielded P. citrophthora. This indicated that run-off water can introduce Phytophthora zoospores from citrus groves into the canals.

In 6 of the 12 canals tested copper sulfate was used for the control of algae and moss; one canal received only paint thinner (a petroleum distillate); commercial aquatic weed killer was added to another; another was given aquatic weed killer plus copper sulfate; and two received no treatment. Most of the chemical treatments were applied during the warm summer months when the ecological factors are conducive to growth of algae and moss. The frequency and amount of application were governed by the quantity of aquatic weeds developing in the canals. No correlation between presence or absence of Phytophthora spp. and the time and kind of chemical application was found.

One of three reservoirs tested during the year was infested with P. parasitica. However, following monthly applications of copper sulfate to the infested water, no Phytophthora spp. were obtained despite repeated trapping. This seems to indicate that copper sulfate is effective against Phytophthora fungi under the static condition of the water in a reservoir. Preliminary experiments have indicated that chlorination may be an effective means of disinfesting water of Phytophthora spp. Two parts per million (2.0 ppm) of chlorine maintained in water for 2 minutes kills the infective zoospores.

DISCUSSION

This survey of 15 water supplies for the presence of Phytophthora spp. has revealed the following: (a) 12 canals are capable of infesting with Phytophthora spp. pathogenic to citrus all the 40,000 acres of orchards using these sources of water; (b) any new citrus plantings on land virgin to citrus crops will become infested with Phytophthora spp. when they are irrigated with water from these canals; (c) any old citrus planting sites which have been fumigated with the recommended chemicals to rid them of Phytophthora spp. are subject to reinfestation through use of contaminated water; (d) Phytophthora-free irrigation water can become infested when the open canal traverses infested citrus groves from which run-off water is permitted to drain into the canal; (e) irrigation with Phytophthora-infested water rapidly distributes the fungi in a grove (8); (f) the Phytophthora species existing in canals vary with seasonal temperature changes in the canal water; (g) the lemon-trap method used over a sufficient time period is a reliable technique for testing irrigation water qualitatively for Phytophthora spp. that infect citrus.

Since this survey has shown that Phytophthora fungi are spread widely by irrigation, it may be concluded that irrigation water should be disinfested before it is used by growers. This, however, is difficult and expensive to accomplish. Growers should carefully maintain good cultural practices. These include: providing good drainage, avoiding excessive watering and fertilizing, making use of the more resistant rootstocks which should be budded high and planted high so that the bud union is 6 inches or more above ground level; promptly excising gummosis lesions that develop on trunk and crown and treating the wounds with a disinfectant. Trees even with some infected roots, if thus treated, will continue to outgrow the attack by the fungi and produce at a profit (2, 3, 7).

Finally, the grower should also be reminded that while the use of Phytophthora-free water assures freedom from infection from that source, disinfection of possibly contaminated citrus

seed by immersion in water at 50° C for 4 minutes (6), and planting only in seed beds, nursery areas, and orchard soil disinfested with effective fumigants are also required to supply the most desirable conditions for growing citrus (4).

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A TECHNIQUE FOR DETERMINING THE FUNGICIDAL VALUE OF VARIOUS TREATMENTS AGAINST PHYTOPHTHORA SPP.

L. J. Klotz and T. A. DeWolfe

In determining the thermal death periods and toxicity of chemicals to the zoospores of Phytophthora spp., recording of percentage germination following treatment is confused by germination of the zoospores that took place before treatment. Where a complete kill of the infective spores by a treatment is the main objective we have employed a technique that eliminates the possibility of this misinterpretation.

For testing the effect of temperature, suspensions of zoospores in water in thin-walled capillary glass tubes are exposed for chosen periods of time to the temperature obtained in a water bath. The treated spores are then blown into a cylindrical hole made with a cork borer in an apple and the hole stoppered with the outer half of the removed apple plug. Within a week viable spores produce a typical, firm, brown rot from which the causal Phytophthora sp. can be isolated on an agar medium for identification. Tucker[1] found that of 150 isolates of Phytophthora spp., including 23 different species, all but six isolates, including four species, grew well in apple fruits.

In assaying the toxicity of chemicals, uniform volumes of a zoospore suspension are placed on glass slides and drops of the same volume of various concentrations of a chemical in water mixed with the fungus. After chosen periods of exposure the treated spores are washed with a fine stream from a plastic water bottle into the apple and allowed to incubate as described above.

The use of this method in determining the toxicity of chlorinated water to the zoospores of P. parasitica is shown in Table 1. The period of exposure was the time elapsed from the moment of mixing the chlorinated water with the zoospore suspension to the time the mixture was washed into the hole in the apple.

Table 1. Time required for various concentrations of chlorine in water to kill zoospores of Phytophthora parasitica.

Exposure (in seconds)	Chlorine in water (ppm)				
	0	0.5	1.0	2.0	4.0
10	+[a]	+	+	+	-
20	+	+	+	-	-
40	+	+	-	+	-
60	+	-	+	+	-
120	+	+	+	-	-
300	+	+	+	-	-

[a] + = fungus recovered; - = fungus not recovered from apple.

The minimum lethal exposure to 2 ppm of the chlorine in water was 2 minutes. Where action of the chlorine was not stopped by dilution with water and contact with the apple tissue, a concentration of 0.5 ppm was found lethal to the zoospores.

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[1] Tucker, C. M. 1931. Taxonomy of the genus Phytophthora de Bary. Missouri Agr. Res. Bull. 153. 208 pp.

BRIEF NOTES ON PLANT DISEASE OCCURRENCE

APHANOMYCES RAPHANI ON RED GLOBE RADISH IN CONNECTICUT

By Saul Rich

Red globe radishes on a number of farms in the New Haven area suffered light to moderate damage from black-root disease caused by Aphanomyces raphani[1]. Metallic-gray to black lesions were located mainly at the lower end of the edible root. A number of specimens were badly constricted and cracked. Contrary to the usual symptom picture, there were lesions on the long tap roots. The interior flesh of severely infected radishes showed dark streaks and gray discoloration. Thick, hyaline, non-septate mycelium grew out of sections of diseased tissue kept in a moist chamber overnight. Within 2 days this mycelium produced the clumps of round zoospores typical of A. raphani[2].

Black root, first found in Connecticut in 1931[3], usually is considered an important disease of long white radishes rather than red globe types.

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[1] Kendrick, J. B. 1927. Radish black-root caused by Aphanomyces raphani n. sp. Phytopathology 17: 43.

[2] Kendrick, J. B. 1927. The black-root disease of radish. Purdue University Agr. Exp. Sta. Bull. 311: 1-32.

[3] Clinton, G. P. 1934. Plant pest handbook for Connecticut. II. Diseases and injuries. Connecticut Agr. Exp. Sta. Bull. 358: 153-329.

ANNUAL RYEGRASS, LOLIUM MULTIFLORUM, HOST FOR SCLEROTIUM ROLFSII[1]

By Homer D. Wells[2]

During the past 6 years, many spaced plants of annual ryegrass, Lolium multiflorum Lam., in nurseries at Tifton, Georgia were killed by Sclerotium rolfsii Sacc., which incited a basal sheath and stalk rot. Domestic annual ryegrass and several selections appear to be equally susceptible. This appears to be the first record of Sclerotium rolfsii causing such a disease of annual ryegrass.

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[1] Cooperative investigations at Tifton, Georgia of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the University of Georgia, College of Agriculture Experiment Stations.

[2] Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the University of Georgia, College of Agriculture Experiment Stations, Tifton, Georgia.

ASCOCHYTA HORDEI ON BARLEY IN OHIO

By C. W. Ellett

A leaf spot of cultivated winter barley, caused by a species of Ascochyta, was found during April and May 1959 on University Farm. The Ascochyta was identified by Dr. Roderick Sprague as A. hordei Hara. This is the first report of A. hordei in Ohio. According to Dr. Sprague the only other collections of this fungus in the United States are from West Virginia and the Far West.

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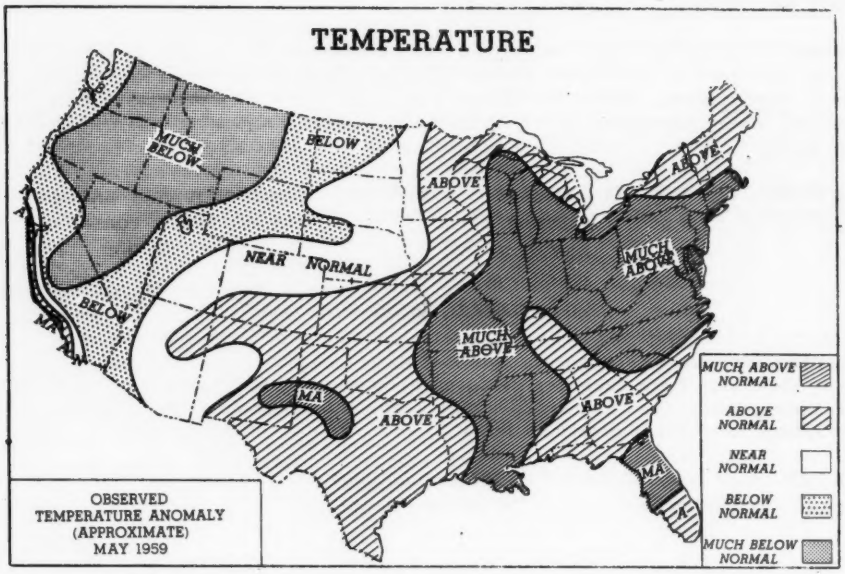
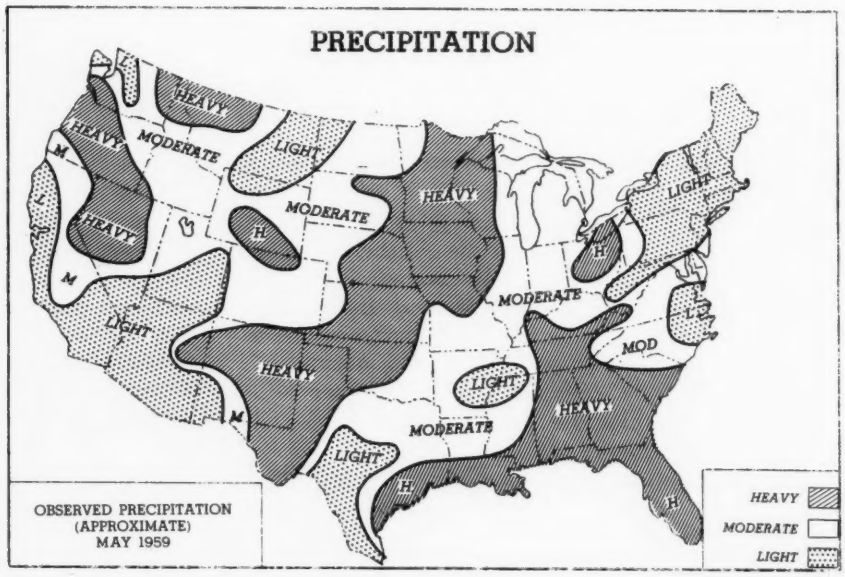
CORRECTIONS

REPORTER, May issue (Volume 43, Number 5).
In the article "Reaction of rye varieties to leaf rust," pages 578-579, the author would like to make the following correction: The decimal points should have been left out of the two tables under the headings "Percent of plants resistant."

REPORTER, June issue (Volume 43, Number 6), page 641: photographs should be interchanged. The REPORTER regrets the error.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.